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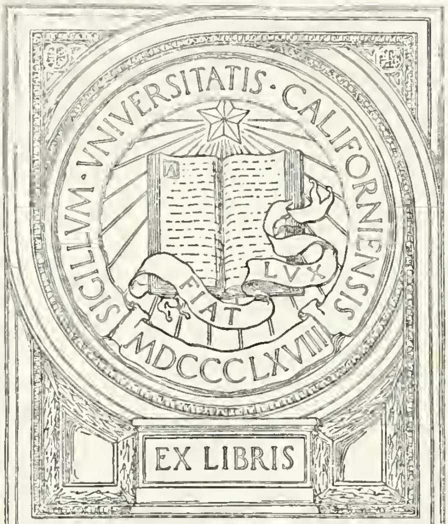

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THE TREATMENT OF SOME COMMON DERMATOSES OF INFANCY AND CHILDHOOD*

JAMES W. BURKS, JR., M. D.
NEW ORLEANS

The treatment of dermatologic diseases in young patients is not difficult if certain fundamental differences in the skin of the child and the adult are kept in mind. These exist especially in the metabolism of proteins, fats, and minerals, and in certain endocrine relations. The chemical composition differs particularly in sodium and potassium ratio and in water content. Nervous and physical reactions are dissimilar. The skin of the child is less oily, possesses less developed hair, less pigment, different secretions, and a more extensive and superficial lymphatic system. Cutaneous reactions during childhood tend to be vesicular, whereas in adults they are more apt to be papular, lichenified, and drier. Immunologic responses also differ.

The delicate, tender skin of the child is more adversely influenced by external factors. Uncleanliness leads to infection; contact with soiled diapers induces irritation. Maternal over-enthusiasm for cleanliness may also produce irritation and excess dryness through frequent bathing, use of strong soaps, medicated oils, and powders. Many mild cutaneous reactions will subside spontaneously if mothers can only be made to realize that every single minor skin abnormality does not require active treatment.

As in all medical practice, effective therapeutics in pediatric dermatology depends upon correct diagnosis. Prophylaxis is a prime consideration. Care of the skin should not be delegated to the whims of parents or to neighborly advice but should be based on sound medical knowledge. The first essential is cleanliness, attained without disturbing the physiologic and chemical condition of the skin. Oily skin can withstand more frequent washings and stronger (alkaline) soaps than dry skin. Ichthyotic skin requires bathing with oil; seborrheic skin tolerates frequent bathing. Scented oils and soaps are apt to please the mother but will displease the dermatologist, who often is faced with treating resulting sensitizations. Some skins are so delicate that they are irritated by plain water; these may be bathed with isotonic saline solution, nonmedicated (oily) preparations, such as equal parts of olive oil and lime water, or colloidal baths. The latter may be prepared with cornstarch and soda.

Colloidal baths

1. Make a cold water paste of:
    1 cup cornstarch
    ¼ cup baking soda
    and put into about
    ½ tub warm water
   Bathe twenty to thirty minutes, daily, or less often if the skin continues to be dry.
2. Boil 2 cups bulk oatmeal in 1 quart water for 30-45 minutes in a double boiler. Allow to cool for 15 minutes, then add ½ cup baking soda. Pour the

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*Read at the Post-Graduate Course in Pediatrics, Tulane University of Louisiana, New Orleans, Louisiana, December 12, 1945.

mixture into gauze bag and tie shut.
Place in a bathtub one-half to three-fourths full of water at about 90-96 F. Patient may remain in tub one-half to two hours, expressing the oatmeal mash through the gauze and applying it to the body. Wash off the mash thoroughly before leaving the tub. Dry by gentle patting, *not* rubbing.

These colloidal baths are excellent in cases of generalized iritations, where soothing measures are indicated. Prolonged use may cause excessive dryness of the skin, which can be corrected by application of olive oil and lime water in equal parts, after bathing. Any attempt to replace natural body oils will be only partially successful at best. Lanolin and aquaphor in various proportions are widely used for lubrication. Too frequent application of oils and ointments to the hairy parts, especially if combined with rubbing, may result in folliculitis.

In general, edematous, erythematous, and exudative eruptions respond to moisture, attained either by wet dressings or baths. After subsidence of the acute process, various powders or lotions may be used. Plain calamine lotion is a convenient way to apply powder to the skin. If this is too drying, a thin coat of a mildly soothing ointment may be applied over the lotion.

An acceptable ointment is:
Boric acid 1.5 (gr. xxii)
Ung. Aq. Rosae q.s. 60.0 (oz. ii)

If the dermatitis has progressed beyond the subacute stage and the skin has become drier, a mildly stimulating mixture may be tried, such as ichthyol, 1 to 3 per cent, in zinc oxide ointment. More stimulating measures should follow, as treatment is tolerated, until complete involution. An excellent keratoplastic is crude coal tar ointment in strength varying from 0.5 to 2 per cent. Exposure to ultra-violet light, carefully increased as tolerated, is highly recommended for more chronic stages.

Treatment must be altered not only for the individual patient but also for different anatomic sites. Ointments should not be employed in intertriginous dermatitis venenata if used in the natural body folds. Ointment applied during the heat of the day rapidly melts, becomes messy, and fast loses therapeutic efficiency. Therefore, the general rule should be to avoid ointments in intertriginous areas and on most cutaneous surfaces during hot weather; lotions, such as calamine lotion, may be substituted. If further antipruritic effect other than drying is desired, 0.5 per cent phenol or 0.25 per cent menthol may be added. Addition of 0.5 to 1.0 per cent ichthyol will produce a mild keratoplastic and some parasiticidal effect.

Treatment of pruritus is necessary not only for relief to the child (and mother) but also for prophylactic reasons. Scratching leads all too frequently to pyoderma. Excoriations are tell-tale signs of pruritus in an uncommunicative patient. Dryness of the skin (asteatosis, xerosis) is probably the most common cause of pruritus. If drying lotions are employed, aggravation will of course ensue. Equal parts of olive oil added to the lotion enhances the soothing effect.

Another useful prescription is:

Menthol	0.3	(gr. v)
Phenol	2.0	(m. xxx)
Olive oil	120.0	(oz. iv)
Lime water	120.0	(oz. iv)
Pulverized zinc oxide	20.0	(dr. v)

SCABIES

Scabies is not diagnosed often in private practice, but many cases go unrecognized. This in part is due to the fact that the disease is ordinarily associated with poor hygiene among underprivileged classes; scabies, like syphilis, however, has no respect for economic or social status. Secondary impetiginous infections increase the importance of this disease in pediatric dermatology. The predominating pyogenic aspects of secondarily infected scabies often obscure the disease. Treatment of scabies itself must often be delayed until secondary infection has subsided. Inefficacy of treatment is usually caused by one of three factors: lack of consideration of reinfection, failure to follow in detail the recommended treatment of the entire skin, or improper disinfestation of clothes and bedding. Parents and siblings should be examined for signs of the disease; if free, they should be isolated and examined periodically until the entire household has been cured. If such examinations are impractical, it is best to treat the entire family.

Although the medical profession is constantly besieged with new, highly publicized scabeticides. the old stand-by, sulphur ointment, still seems to be the most useful and dependable remedy. Variations of the following prescription may be used:

Sulphur precipitate	6.0	(gr. xc)
Balsam Peru	6.0	(gr. xc)
Petrolatum		
Lanolin aa q.s. ad	120.0	(oz. iv.)

Efficacy of treatment depends on detailed attention in its application. Printed forms like the following may be useful:

Observe these rules carefully, and do not repeat the course without your physician's permission, or you may irritate the skin.

First night: A cleansing bath with soap and water to all parts of the body. Follow this with liberal application of the ointment to the skin from the neck downward, including the folds of the body and the regions between the fingers and toes.

Second day: Apply ointment again morning and night under the same clothing. No bath is given.

Third morning: On the following day, or about twelve hours after the third application of ointment, cleanse the skin thoroughly again with a warm soap and water bath. This completes the course.

Clean underwear and bed linen must be used from this point on. It is not necessary to destroy the old clothing; thorough laundering or dry cleaning will do the work. The mite lives not in the clothing but in the skin and is transmitted from one individual to another by close personal contact.

After such a course of treatment, if itching persists, some soothing lotion (such as equal parts of olive oil and lime water) should be applied to the skin. If another course of treatment seems indicated, your doctor should be consulted.

For very delicate, tender skin, a lower concentration of sulphur should be used. Balsam of Peru alone in a petrolatum base may suffice if applied after thorough bathing with soap and water. For infants, a useful prescriptions is:

Storax (USP)	6.0	(gr. xc)
Balsam Peru	6.0	(gr. xc)
Soft white paraffin q.s.	30.0	(oz. i)

Benzyl benzoate is favored by some practitioners. Its use on excoriated scabetic skins often produces undesirable irritation. If it must be employed, treat the inflammatory complicating condition first. In general, this scabeticide is more valuable in treatment of adults. Wash cloths impregnated with sulphur have been used advantageously.

It must be remembered that sulphur is capable of producing a serious dermatitis which may obscure the progress of the disease and produce a condition very resistant to treatment. In such cases of sensitization, after subsidence of the complicating dermatitis, ichthyol in 1 or 2 per cent strength may usually be substituted safely. Pyrethrum also may be used, but it is not an uncommon sensitizer. If sensitization or irritation develops, any energetic scabetic regimen should be abandoned and soothing measures instituted, in the form of daily colloidal baths, followed by application of lotions containing phenol or menthol in an oily vehicle. With subsidence of the complicating irritation, one of the above mentioned alternative measures may be employed. The application of sulphur to the face may irritate the eyes; the very young patient may inadvertently carry sulphur to the eyes by rubbing. If extensive application of the ointment to the hands is necessary, a splint restraint for the arms should be used.

PYODERMAS

Immunity to a number of diseases is transmitted from the mother, but immunity to pyogenic cutaneous disease is a notable exception. For this reason, pyodermas in infants and young children are often serious and difficult to control. Contact with excreta, exposure to insects, excoriations from scratching or chapping of the skin through improper cleansing, all provide portals of entry for bacteria. The extensive and superficial lymphatics sometimes fail to cope with infection of the skin. Periumbilical infections in infants may have disastrous results.

Impetigo in infancy is a serious disease which may even be fatal. It is highly contagious and spreads rapidly in institutions. Its chronicity, recurrence, and refractiveness to treatment are often embarrassing to the practitioner. Measures that cure impetigo in one patient may fail miserably in another. Ammoniated mercury seems to be more helpful in staphylococcal types, gentian violet in streptococcal. Clinical differentiation may be summarized as follows: The staphylococcal type occurs most frequently in July. The primary lesion is a large vesicle without erythema, which may attain a diameter of 5 cm. It tends to be circinate and to form crusts, is usually of a longer duration and less contagious than the streptococcal type. The latter has a higher incidence in the fall; its primary lesion is of pinhead size and ruptures early. There is a broad erythematous areola, which forms thick yellow crusts; it is extremely contagious but of a shorter duration than that due to staphylococcus.

Ammoniated mercury ointment has withstood the test of time so well that it should be tried first in the treatment of impetigo. A 5 per cent strength is adequate if the drug is going to benefit at all; a stronger percentage will only be irritating. After removal of the non-penetrable crusts, the ointment should be rubbed into the lesions with special attention to the active border. Bandaging with loose mesh gauze but avoiding too much occlusion is advisable. Adhesive tape should not be applied directly to the skin of a patient with impetigo because trauma from removal of the tape spreads the disease. The routine is to apply the ointment at night, remove the crusts in the morning by soaking with soapy water or using wet dressing of mercury bichloride (1:4000). The crusts should be removed as gently as possible: impetigo rarely produces scarring except by excoriations or injudicious treatment. During the day it is customary to use a drying antiseptic preparation. A 1 to 2 per cent aqueous solution of gentian violet is advocated by some, but others object to its staining properties. A currently popular and efficacious product is the alulotion of ammoniated mercury. For infants, this may be diluted with equal parts of Kaomagma and painted on the skin twice a day. If impetigo complicates scabies, prescriptions containing sulphur are desirable. Very extensive and exudative impetiginous patches frequently respond to wet dressings of bichloride of mercury in dilution of 1:4000, applied either continuously or for an hour or more several times daily.

The sulfonamides have been used indiscriminately in treatment of the pyodermas. In view of the serious nature of impetigo in the infant, there is some justification for its use in cases of rapid, extensive involvement. For the few lesions of early impetigo, the previously mentioned measures should suffice; sulfonamides should be reserved for resistant or fulminating cases. It is hardly justifiable to chance sensitization of a very young patient to a valuable lifesaving drug through treatment of a minor skin condition which ordinarily responds to less innocuous measures. Generalized systemic or local sensitization may occur from topical use of these drugs. Sulfonamide rashes may persist for months, preventing valuable exposure to ultraviolet light because of danger of photosensitization. If effective at all, local administration of sulfonamides will produce results in three to ten days. A strength of 5 to 10 per cent may be used in a vanishing cream base or surgical lubricating jelly. Recently, some authors have reported better results with higher concentrations.

The exact effectiveness of penicillin in pyodermas is not known, but many favorable results have been reported. Less dangerous than the sulfonamides, it is the drug of choice when more than local treatment is indicated. Early administration of 5000 to 10,000 units intramuscularly every two to three hours for as many days should be tried in resistant cases. Fulminating conditions require greater amounts over longer periods. Oral administration of penicillin may be tried for the ambulatory patient. The initial dose is often given intramuscularly in order to raise the blood level rapidly. Maintenance of this level may be attempted orally with two to three times the parenteral dosage. Another plan consists in giving the drug intramuscularly during the day and orally at night. In some clinics penicillin has almost completely replaced former therapeutic measures for impetigo. Locally, the drug may be prescribed in a water soluble base containing 400 to 1000 Oxford units per gram. Deterioration can be reduced by dispensing small amounts and storing at low temperatures. Wet dressings of saline solution in similar concentrations are useful. Economy may be secured through use of washings recovered from discarded ampules. A few reactions to penicillin are known to occur (toxic erythemas, vesiculation, and urticaria), most of which are so mild that treatment need not be interrupted.

MILIARIA
"HEAT RASH"

The nuisance value of miliaria is great; frequently it leads to more serious diseases, such as impetigo and furunculosis, especially in infancy. Little is known about its cause, but it is obviously associated with excessive sweating. Only recently has the physiology of sweating been investigated. Limited pathologic studies have contributed little. Some blame yeast or fungi; others attribute irritation to the perspiration itself. The disease does not seem to be associated with filth *per se*. Unfortunately, the laity ascribes it to infrequent bathing and bathe even more often and more energetically, thereby aggravating the basic condition.

The following concept of the disorder is an interesting one: A fundamental function of the oil of the skin is to protect it from sweat. In hot climates the sebaceous glands are stimulated to protect the skin from sweat. Physiologic imbalance of oil and sweat production in certain individuals may then be responsible for the eruption. Natives of the tropics seem to have a perfect balance of these mechanisms.

Sudamina, a non-inflammatory engorgement of the coil glands, is apparently due entirely to excessive sweating, is more physiologic than pathologic, and requires no treatment. Since the minute vesicles of the disease are superficial, secondary infection need not be feared.

For miliaria, tanning of the skin is often a beneficial prophylactic and even remedial measure. Physical exertion during exposure to the rays of the sun seems detrimental, whereas deliberate gradual exposure for short periods of time may help. In other words, if exposure to sun results in excessive production of sweat, no benefit will be obtained. Bathing should be curtailed, if not completely abolished; certainly long baths, use of hot water and strong soaps should be discouraged. Colloidal baths can easily be substituted. Liberal amounts of cooling fluids should be taken and extra salt added to the diet. Hot fliuds and foods, as well as highly seasoned ones, should be avoided. Sweating and irritation should be minimized by all possible means. In unclothed infants sweat should be removed by blotting the skin with soft absorbent cloths, followed by the application of oil. Obesity should be corrected. Removal of sweat may be secured through powders which induce evaporation by increasing surface area. This is a more desirable method, since exposure to draughts with resultant drying leaves irritating chemical and cellular debris behind. A suitable powder is:

Zinc oxide
Zinc stearate
Talcum aa

This may be used with or without the addition of 4 to 6 per cent camphor. Calamine lotion is convenient. Another less desirable formula is:

Witch hazel

Saturated solution boric acid aa

The use of highly medicated proprietary preparations in the treatment of heat rash is not advisable. The average mother is reticent about consulting her physician for treatment of a simple heat rash; consequently, it is only after a complicating dermatitis venenata or pyoderma develops that she usually seeks aid. Alcohol, through local anesthetic properties, gives temporary relief, but ultimately its drying effect may aggravate the basic condition.

DERMATITIS VENENATA

Treatment of dermatitis venenata is more successful than that of any other group of skin diseases. Success depends largely on early diagnosis, removal of cause, and simple soothing measures.

When an infant starts moving about, the number of possible irritants he may contact increases sharply. A classical example of disease due to external causes is Rhus dermatitis or poison ivy. Dermatitis venenata deserves proper care if for no other reason than avoidance of secondary infection.

Scrubbing with soap and water immediately after contact will remove much of the irritant and thus partially reduce severity of symptoms. Following this, a soothing wet dressing should be applied. Liquor aluminum subacetate (Burrow's solution), in a 0.5 per cent solution, or saturate solution of boric acid may be used. Coop preparations seem most comforting. A roller type dressing of eight to ten thicknesses of wide mesh gauze should be permitted to remain in contact with the skin as long as it is wet. Before completely drying, it should be removed from the affected part, dipped in solution, and reapplied. Pouring the solution over the compresses results in concentration and further irritation. By removing, rinsing, and changing gauze, excreta and debris from the area will be carried away. Undue maceration may be prevented by allowing the skin to dry for about fifteen minutes every two to three hours. Frequent or continuous collodial baths are also indicated.

There is some controversy as to whether or not vesicles and bullae should be opened. In climates where pyodermas are prevalent, the vesicles should be opened in an attempt to prevent secondary infection; otherwise bacteria in the superficial layers of the skin find an excellent culture media in the contained serum. If infection should develop, debridement is essential. For complicating infections a good wet dressing consists of 1:12,000 potassium permanganate; this is soothing, drying, and antiseptic. When involvement is extensive, baths with this solution in a dilution of 1:20,000 are helpful. Weak solutions of bichloride of mercury or silver nitrate may also be used.

Desensitization either with elixir of the Rhus product or by parenteral administration of the oily substance of the plant should be avoided during the acute stage. Favorable results doubtless occur, but the probability of dissemination makes this practice hazardous at this stage. It should be reserved for prophylaxis prior to anticipated contact, and even here its value is questionable. The favorite home remedy of chewing the leaves may have obvious disastrous results! Recent attempts at oral desensitization by use of capsules containing the oil have possibly effected some benefit but have also produced a number of instances of perianal dermatitis venenata. A familiar complication of an old, well accepted therapeutic measure is the tattooing ensuing from the use of iron-containing preparations. The best advice a practitioner can conscientiously give a sufferer is: "Leaves three, let it be."

Any discussion of this group of diseases must include a common, readily avoidable and easily treated one, ammoniacal dermatitis. Urea in the urine decomposes into ammonia by action of saprophytic bacilli from the stool, giving rise to a characteristic smell and irritative dermatitis. Use of coarse, rough, improperly laundered diapers sets the stage. Consequences may be so severe as to cause retention of urine either by occlusion of the urethral meatus or voluntary withholding of urine because of pain. Prophylaxis consists of avoidance of overfeeding and resultant diarrheas, frequent diapering with soft clean material, and proper laundering. The diapers should be finally rinsed in a saturated solution of boric acid. Drying lotions, dusting powders, and oil inunctions are also beneficial.

LANATOSIDE C (CEDILANID)

S. P. WISE, III, M. D.
NEW ORLEANS

As William Withering, the father of digitalis therapy, said many years ago, "It is much easier to write upon a disease than upon a remedy. The former is in the hands of nature, and a faithful observer, with an eye of tolerable judgment cannot fail to delineate a likeness. The latter will ever be subject to the whims, the inaccuracies and the blunders of mankind." By way of definition, however, it may be stated that cedilanid is a pure, stable, crystalline, adequately soluble, constant in action, lanatoside C, one of the principal clycosides of Digitalis lanata. Cedilanid is the trade name for this preparation. It must be added that this glycoside is not present in Digitalis purpurea.

The purpose of this paper is to discuss briefly the following points: (1) The chemistry of the cardiac glycosides, with special reference to cedilanid; (2) the cardiovascular actions of cedilanid and comparison with some of the other glycosides; (3) the routes of administration, their indications and dosage; (4) some aspects of toxicity.

CHEMISTRY

Medicine is deeply indebted to Stoll[1] and his co-workers for the brilliant work regarding the chemistry of the cardiac glycosides. Previous to the work of this author, much confusion was present regarding the true chemical nature of these substances. Stoll defines the "genuine glycosides" as "the unchanged initial cardiac glycosides as they pre-exist in the fresh plant, carefully dried," and consist of an aglucone nucleus linked by a side chain to a various number of sugars. The glycosides as previously known were shown to be degeneration products of the genuine glycosides, some of the sugar molecules having been lost. This author, using a special process, was able to extract the genuine glycoside complex, Digilanid, from which the pure crystalline genuine glycosides, the lanatosides A, B, and C were subsequently extracted. These glycosides can be determined gravimetrically, thus obviating the necessity for bio-assay, as well as containing no enzymes so that no change in potency results from storage.

Further, Stoll[1] found in studies on Digitalis purpurea two amorphous initial glycosides chemically related to lanatosides A and B, corresponding to the previously known digitoxin and gitoxin respectively, the latter being deficient in a glucose molecule. On the other hand, lanatoside C, corresponding to digoxin in a similar manner has no chemical counterpart in the glycosides of Digitalis purpurea. Thus it is seen that lanatoside C is a genuine glycoside differing chemically from all other known glycosides, and existing in a crystalline state, and hence stable, adequately soluble, and constant in action. (See Fig. I for interrelationships of the glycosides.)

Chen and co-workers[2] state that the removal of a sugar from the glycoside molecule diminishes the persistence of action of the drug. They state further that each aglycone has a less powerful effect on the heart than its parent glycoside. Also it was found that the aglycone had a higher emetic action than the parent glycosides.

Gold[3] disagrees diametrically with Chen and using the human assay method found that:

(1) Digitoxin (Digitaline nativelle) was ten times and gitalin four times more potent than digitalis leaf or cedilanid.

(2) As to absorpition the ratio between the oral and intravenous dose is ten to one for cedilanid, one to one for digitoxin, and five to one for digifoline. It must be remembered, however, that here rate of destruction and elimination is included. Further, Sokolow and Chamberlain[4] on the other hand found this ratio of oral to intravenous dose to be only 1 to 2.8. Dille and Whatmore[5] in a series of experiments in which cats with isolated loops of stomach, intestine (ileum and jejunum) and colon with

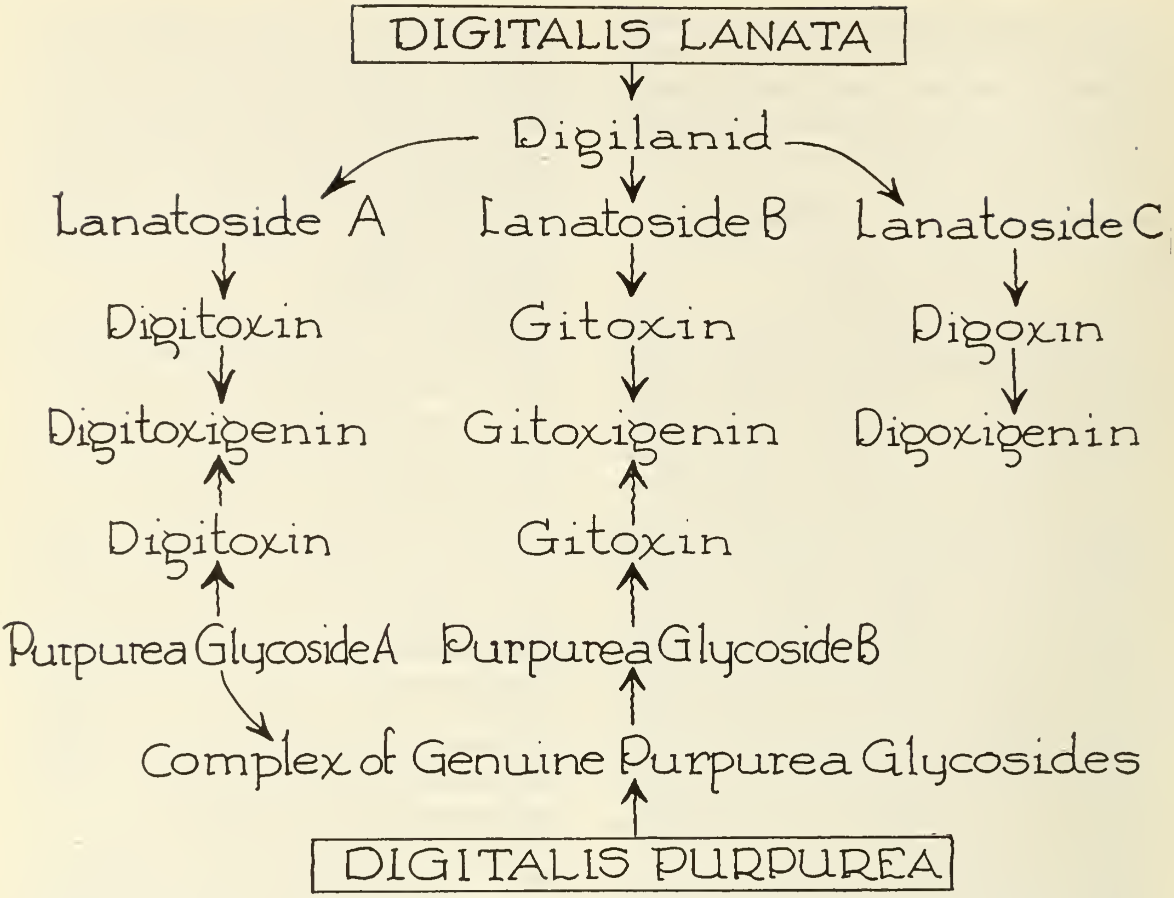

Fig. 1. Chemical relationships between the glycosides of Digitalis lanata and purpurae (modified from Sokolow and Chamberlain[4] after Stoll and Kreis).

their blood supplies intact were used, cedilanid and tincture of Digitalis U.S.P. XI were injected into the lumen; after one hour the loop of gut was removed and bioassayed on frogs while the cats received intravenous infusions containing the same drug to point of death.

By this method they found greatest absorption of both drugs from intestine (about three times as much as from stomach or colon.) Also cedilanid was found to be absorbed about three times as rapidly from the intestine as tincture of digitalis (determined by varying the length of absorption.)

Discrepancies were found between the calculated essential absorption and the apparent absorption which is indicative of partial destruction or inactivation of both drugs in the lumen of the gastrointestinal tract by digestive enzymes.

(3) As to speed of action Gold[3] found that: (a) the oral action varies with speed of absorption and: (b) that by means of intravenous administration ouabain digitalizes the patient within two hours, digitoxin in six hours, with cedilanid falling somewhere between the two. Fahr and LaDue[6] found that cedilanid administered intravenously to patients with auricular fibrillation reduced the heart rate to normal within a period of from two minutes to two hours, and when administered orally within 24 to 48 hours.

(4) Concerning speed of elimination, Gold[3] states that ouabin and cedilanid have about the same rate, that is, less than two-thirds of the effect being lost in four days, whereas with digitoxin less than one-third of the effect is lost in four days.

DeGraff and Lehman[7] have pointed out

that there may be some qualitative difference between various cardiac glycosides. They state, "The lethal effect of lanatoside C dissipates rapidly during the first 24 hours after intravenous injection in the cat. The lethality of digitoxin, however, actually increases during the first three hours and is still quite strong after 24 hours. This very considerable qualitative difference between the action of the genuine glycoside and the product of its partial hydrolytic degradation appears not to have been emphasized before. As would be expected, the effect of digoxigenin completely disappears in 24 hours. At three hours, however, digoxigenin causes an increased tolerance for ouabin. Further investigation is needed to determine the significance of this finding." The interesting, if somewhat confusing, conclusions of these authors may, however, point the way for future investigation along this line.

CARDIOVASCULAR ACTIONS

It has long been recognized that the primary and most important action of the cardiac glycoside is that of increasing the force of the systolic contractions of the heart. Gold[8] using the papillary muscle of the cat performed a series of experiments in which all the secondary factors which may affect the diastolic heart size were removed. It was found that digitalis does not influence the length of the resting fiber, but merely causes increased force of contractions. Investigating the theory that Starling's law was effective up to a certain point above which over-distention occurs, the author, using cats, occluded the aorta and caused intra-auricular pressure to rise to 100 cm. of water. However, this failing and distended heart thus produced still obeyed the normal law, that is, with a lowering of venous pressure, the force of contraction declined and with a raising of the venous pressure, the force increased. Studies by others in man exhibit the same so that there is no reason for assuming that in clinical heart failure the muscle is overstretched or that making the muscle smaller with digitalis will improve its function. Thus it is seen that the decrease in heart size is entirely secondary to improved systolic force rather than being its cause. These experiments may be criticized because of the absence of physiologic conditions, as well as the fact that myocardium from experimental animals rather than humans was studied. LaDue and Fahr[9], believing with Cohn and Stewart that the decrease in diastolic heart size was important, studied this action in a group of patients and reached the following conclusions which are quoted directly because of their importance and clarity of meaning.

"1. The intravenous administration of lanatoside C. to patients with heart failure and normal sinus rhythm usually increases the pulse pressure and reduces the circulation time and venous pressure within two hours. Roentgenkymographic studies of the hearts of ten such patients showed that the drug produced a significant reduction of diastolic heart volume in five patients and an increase of stroke output in eight patients. In two patients, the minute volume of the heart was so large before the administration of the drug that no great increase could be expected.

"2. In the eight patients who responded with increased cardiac output the product of the stroke output and the mean blood pressure was definitely increased, whereas the diastolic heart volume decreased or was unchanged. Since the diastolic heart volume is an index of oxygen consumption, these increases in work which resulted from the administration of lanatoside C. must have represented proportional improvement in the mechanical efficiency of the hearts of eight of the ten patients studied.

"3. In seven patients who were restudied after the establishment of compensation, there was a decrease of 200 c.c. or more in the diastolic heart volume. In four patients, the minute volume was greater than at the start of the experiment, and in three patients it was less.

"4. The most consistent and lasting change in the failing heart that is produced by the administration of lanatoside C. seems to be a decrease in its diastolic volume, or oxygen consumption."

Perhaps a fair evaluation of the work done to date would be that all other important effects of the cardiac glycosides are probably secondary to an increase in the force of systolic contraction and its accompanying improved ventricular emptying.

The mechanism by which digitalis causes a slowing of heart rate has long been a subject of debate. Gold and coworkers,[8, 10] in reviewing the literature on this subject, found that whereas Cushney believed that in auricular fibrillation digitalis slowed the heart by its action directly upon A-V conduction and Lewis thought the action was largely through the vagus, Elsie Porter thought the action was entirely through the vagus. Gold found that in the human both factors are active, with passage from the vagal to the extravagal form of slowing as the drug is increased and in the reverse direction as the drug is eliminated. The vagus is found to be stimulated reflexly as a result of improved myocardial function. Quoting Gold[8], "The problem of control of heart rate in patients with auricular fibrillation, which rises during free physical exercise even though satisfactory 'rest digitalization' is present, has a direct bearing on the vagal-extravagal question. It was found that in patients receiving (1) no digitalis, (2) moderate doses of digitalis, and (3) large doses of digitalis, atropine intravenously in doses sufficient to block the vagus has the same effect in increasing the rate in all three groups as did climbing stairs to the limit of endurance. Digitalization to the extravagal extent prevented this increase in rate, although in some cases the dosage required was quite close to that which causes impaired appetite and nausea."

Gold[10] further found that which of the two factors, that is, vagal or extravagal, predominates is entirely a function of the degree of digitalization and not a matter of individual peculiarity, degree of heart failure, or the length of time the heart has been under digitalis. The maximum effective dose of atropine for determining which factor predominates was found to be gr. 1/30 given intravenously every five to six hours. It is readily seen that the use of atropine to determine the degree to which a patient is digitalized may be of great value, specially when one is using potent intravenous glycosides.

Further discussion as to the effect of digitalis on diastolic heart size, cardiac output, venous pressure, blood pressure and diuresis, in humans, is beyond the scope of this paper. However, the effect of the glycosides upon coronary circulation is of considerable importance. LaDue[11] found that whereas Essex and Visscher had shown that lanatoside C in therapeutic doses does not restrict coronary flow as measured by the thermostromuhr, myocardial damage may be produced in the hearts of dogs by the daily intravenous injections of doses of cedilanid greater than 0.20 cat units per kilogram of body weight. The fact that no gross or microscopic abnormalities were found in the coronary vessels even in the presence of subendocardial hemorrhages in animals who died after these injections points to the probability that cedilanid must exert a powerful vasoconstrictor action on the vessels in large doses. This point will be dealt with further under toxicity.

As to the electrocardiographic changes produced by the various digitalis preparations, it is common knowledge that T wave changes as well as shifts in the S-T segment are always present. An interesting study by Tandowsky[12] revealed that the average time required for the production of maximal RS-T segment changes after the administration of 1.6 mg. of cedilanid intravenously (this being the usual digitalizing dose recommended) was 27.5 minutes in persons with normal hearts as compared with six hours and fifteen minutes, after a single oral dose of 16 grains of Digitalis purpurea. In patients with congestive failure treated similarly intravenous cedilanid produced maximum changes in the RS-T segment in two to three hours whereas oral Digitalis purpurea did so in thirteen to fifteen hours. In the normal control group RS-T changes returned to normal in about sixteen hours after the single intravenous dose of cedilanid and in 48 hours, after the single oral dose of Digitalis purpurea. Tandowsky further observed that with intravenous cedilanid the clinical manifestation of congestive failure disappeared more rapidly than with the oral leaf and that the emetic quality of the leaf was almost entirely absent with intravenous cedilanid.

ROUTES OF ADMINISTRATION, THEIR INDICATIONS, AND DOSAGES

The indications for the oral use of cardiac glycosides are so obvious that they will only be listed:

(1) Congestive heart failure, from any cause whatever, is the true primary indication for digitalis.

(2) Arrhythmias: (a) Auricular fibrillation or flutter with or without heart failure; (b) paroxysmal tachycardia with impending failure.

Whenever the subject of dosage arises in regard to the cardiac glycosides, the question of bio-assay immediately presents itself. Suffice it to say that Gold[8], and his coworkers,[13, 14] have led the way in replacing the frog method of U.S.P. XI with the far more accurate (as to potency in man) cat method of Hatcher and Brody which became official in U.S.P. XII. Possibly nowhere in medicine, however, is the value of gravimetric dosage of a drug more important than in the use of cardiac glycosides. Only through the use of human bio-assay as advocated by Gold and Cattell[13] can the potency of the pure glycosides be determined but once these values have been ascertained the clinician will be relieved of the alarming toxicity which has so frequently manifested itself with the use of crude drugs in the past. The human bio-assay has been criticized as employing unphysiologic criteria (that is, ventricular rate changes in patients with auricular fibrillation and T-wave changes in patients with regular sinus rhythm), but these criteria are certainly the most objective and easily measured effects yet found and Gold[15] has applied his findings to over 1000 patients and shown them to be satisfactory, at least in his own estimation.

The remainder of this section will be limited in extent largely to the discussion of four representative digitalis preparations: the whole leaf of Digitalis purpurea, cedilanid, digitoxin, and ouabain. The principles stated here may be applied to various other preparations, but as most cardiologists agree, the clinician should familiarize himself with a few of the available digitalis preparations and learn what can be expected from their use in order that the danger of becoming lost in the maze of high pressure propaganda and grandiose claims made from time to time by the various drug houses may be obviated. All doses will be stated in the metric system since the greatest universality of measuring and understanding is thus attained.

Using cedilanid orally for digitalization, Fahr and LaDue[6] advise giving 3.75 mg. at once, followed by 2.5 mg. in the next 12 to 24 hours in single or divided dosage. Sokolow and Chamberlain[4] recommend 7.5 mg. given in three days and Gold[15] found that 1.5 to 2.5 mg. daily gave good results. It should be stated that cedilanid is marketed in 0.25 mg. and 0.5 mg. tablets so that ease of administration is facilitated.

Using digitoxin (Digitaline nativelle) Gold[15] in 1000 unselected cases found that 1.2 mg. in a single dose served to digitalize the patients in from six to ten hours with almost no toxic effects. This dose is equivalent to 1.2 gm. of the leaf which requires 24 to 48 hours for digitalization since it cannot be given at one time because the local gastrointestinal irritation will cause nausea or vomiting in 10 to 20 per cent of the cases. An alternate regime using digitoxin is 0.2 mg. every four to six hours if slower action is preferred. The fact that digitoxin is completely absorbed from the gastrointestinal tract, thus making the oral and intravenous dose the same, is a strong argument in favor of the use of this drug for routine oral digitalization in preference to cedilanid which is only one-tenth (Gold[15]) or one-third (Sokolow and Chamberlain[4]) absorbed from the gastrointestinal tract.

As to oral maintenance dosages, an average of 1.5 mg. of cedilanid, 0.1 to 0.2 mg. of digitoxin, or 0.3 gm. of the leaf is required. Ouabain and strophanthin are, of course, given only intravenously.

The indications for intravenous digitalization are as variable as the number of men writing on the subject. LaDue[16] states that in Europe where strophanthin (or ouabain) has been used much more widely than in the United States, the indications are: (1) Acute failure of a high degree, that is, cardiac asthma; (2) slowly progressive failure with acute distress; (3) for patients unable to take digitalis orally. Pick[17] states that intravenous strophanthin is indicated in: (1) Acute cardiac insufficiency due to previously untreated hypertensive disease; (2) mitral stenosis during delivery; (3) coronary diseases developing acute pulmonary edema and nephritis; (4) paroxysmal tachycardia with rapid onset of decompensation.

He states that the only contraindication to intravenous strophanthin therapy is previous digitalization. For the experience of one clinician in the use of ouabain and a comparison of its value with that of digitalis in the treatment of heart failure, the reader is referred to an excellent article by Chavez[30] of Mexico City, in which physicians of the United States are vehemently criticized for having neglected the use of ouabain while it was being used to great advantage in Europe and Latin America. This view is not shared by LaDue[16] who warns that strophanthin is a dangerous drug because of the incidence of toxicity including: (1) uncontrollable nausea and vomiting; (2) heart block; (3) cardiac standstill; (4) ventricular tachycardia, and (5) ventricular standstill. I feel unqualified to evaluate these representative views which are so extremely contradictory.

Pick,[17] however, states that intravenous cedilanid more closely approaches strophanthin as to rapidity of action and weaker cumulation than any of the other available digitalis glycosides and has the added advantages of great therapeutic breadth and a remarkable diuretic action.

Tandowsky[12] writes that intravenous cedilanid is indicated: (1) When the element of time is an important consideration in the treatment of congestive failure, that is, moribund patients; (2) for patients who are suffering from nausea and vomiting when rapid digitalization is indicated; (3) in auricular fibrillation with congestive failure, since he found that three of 11 patients regained a normal rhythm in a group so treated whereas none of the group digitalized with oral leaf regained a normal rhythm.

Investigating the oft-stated contraindication to intravenous digitalization, that is, previous use of oral digitalis within three weeks, Ray and La Due[18] found that the absence of toxic manifestations and the definite improvement evidenced after the intravenous use of cedilanid in patients taking digitalis leaf before and during the onset of failure, suggested several conclusions which are quoted directly. "Since the rate of improvement was more rapid than that to be expected from bed rest alone, it seems obvious that these patients were not receiving adequate therapeutic amounts of digitalis. A combination of factors may explain the tolerance of these patients to the intravenous administration of lanatoside C. The need for more digitalis when heart failure occurs, no matter what the precipitating factor, plus the possibility that the destruction and elimination of the digitalis accumulated during the period of 'digitalization' contributed to the onset of congestive failure.

"From our results, it would seem to be reasonably safe and usually of therapeutic benefit to give 0.4 to 0.8 mg. of lanatoside C intravenously to patients who develop congestive heart failure while taking maintenance doses of from 0.1 to 0.3 gm. of folia digitalis.

"Finally this study indicates that the mere finding of 'digitalis effect' in the electrocardiogram is apparently not proof that the patient is receiving adequate therapeutic doses of the drug.

"1. The intravenous administration of from one-half to the full digitalizing dose of lanatoside C to patients developing congestive heart failure while taking maintenance doses of Digitalis purpurea was followed by definite improvement in forty-six of the sixty-two patients so treated.

2. Objective evidence of improvement was shown by thirty-six of forty-nine patients with regular sinus rhythm and by ten of sixteen patients with auricular fibrillation.

"3. There was only one toxic reaction among the sixty-two patients.

"4. The presence of electrocardiographic evidence of 'digitalis effect' is not proof that therapeutic amounts of digitalis are present in the body (the authors[10] used the atropine test alluded to above).

"5. The mortality of the patients studied was no greater than that previously observed[6] in a group of patients with heart failure of comparable severity who had not been taking digitalis within three weeks prior to intravenous digitalization.

"6. None of the deaths occurring during this investigation could be attributed to the intravenous administration of lanatoside C."

As an addendum to this paper, Ray and LaDue[18] stated, "Since submission of this manuscript, thirty-five patients with sinus rhythm and fifteen patients with auricular fibrillation have been added to the series. There were no toxic manifestations in the patients with auricular fibrillation; six of the patients with regular sinus rhythm developed transient toxic reactions. Thus of 104 patients with regular sinus rhythm, seven, or 6.7 per cent, developed nausea, emesis, and occasional extrasystoles. None of the patients with auricular fibrillation experienced toxic reactions and no deaths could be attributed to the administration of the drug."

This beautiful piece of clinical investigation may be criticized in that no control group was used, but certainly it indicates that many of the alleged accidents and poor results with intravenous cedilanid are mere heresay and not based on scientific medical facts.

If further clinical testimonials as to the value and safety of intravenous cedilanid are desired, the reader is referred to papers by Chamberlain and Sokolow,[19] Sokolow and Chamberlain,[20] Nicholson,[21] Margolin,[22] and Hrenoff.[23]

The dosage by intravenous injection for cedilanid is 1.6 mg. in a single dose, this being 8 cc. of the solution available in 2 cc. and 4 cc. ampules, or an alternative of 0.8 mg. followed in two hours by 0.8 mg. For ouabain the dosage is 0.25 mg. which may be repeated in two hours if required. For digitoxin the intravenous is the same as the oral dose, 1.2 mg. in a single dose.

Intramusclar injection of cedilanid may be used but is often accompanied by local pain and discomfort so that it is seldom recommended or indicated.

Rectal administrations of tincture of digitalis diluted 1:7 or powdered digitalis as suppositories may be used in the same dosage as the oral; cedilanid is not available in suppositories.

With an eye to the future as to some possible "special indications" for cedilanid in preference to other glycosides, the following statements are presented for the readers' evaluation. Several authors,[4, 12, 24] have suggested a relatively high incidence of return to normal rhythm in patients with auricular flutter of fibrillation or supraventricular tachycardia in patients receiving intravenous cedilanid as compared with Digitalis purpurea although no well-controlled studies along this line have been made.

Fahr and LaDue[6] controlled the heart rate and reduced venous pressure from 22 to 9 cm. of water in 48 hours in one patient with auricular fibrillation from hyperthyroidism who failed to respond to the leaf.

Erickson and Fahr[25] advocate the use of cedilanid orally in organically diseased hearts which appear compensated when the circulation time by the calcium gluconate arm-to-mouth method is greater than 16 seconds. This regimen is reputed to do no harm and is beneficial to many cases with circulation times of less than 16 seconds, and it is felt by these authors that life expectancy may be increased as well as physical fitness and that congestive failure may be prevented for a period of years.

Tandowsky[26] suggests that paroxysmal auricular arrhythmias may be stopped by intravenous cedilanid and then the drug may be used orally in doses of 0.5 to 1.0 mg. daily (varying with electrocardiographic effects) as a prophylactic against recurrence of the paroxysms.

TOXICITY

Since this subject has been touched upon many times earlier in this presentation, only brief additions will be made. The toxic effects of strophanthin have already been listed and those for the leaf are familiar to all, that is, nausea and vomiting, diarrhea, and almost any known clinical cardiac arrhythmia with "coupling" or bigeminy and extrasystoles seen most frequently.

Cedilanid has been shown to produce most of the toxic manifestations. Whereas Moe and Visscher[27] concluded that lanatoside C had thirty times as great a ratio of the toxic to therapeutic dose as lanatoside B, they were, as was pointed out by Gold and Cattell[13] comparing concentration in heart-lung preparations to the fatal intravenous dose in cats. Comparing ouabain, digitoxin and the three lanatosides (A, B, and C) by the papillary muscle technic, Gold and Cattell[13] found the toxic to therapeutic ratios about the same for all; and using oral administration to man the percentage incidence of toxicity for full therapeutic and double therapeutic doses of leaf, digitoxin and cedilanid were found to be the same for all three.

LaDue's studies[11] on coronary spasm in dogs caused by massive doses of cedilanid intravenously have been described. In a later article LaDue[28] reported that whereas prolonged daily oral administration of leaf or cedilanid in doses one and a half to ten times those given for clinical maintenance produced no morphologic changes in the hearts of dogs; daily intravenous doses of 0.2 mg. or more of cedilanid per kilogram of body weight gave necrosis, fibrosis and atrophy of the myocardium in 44 per cent of the dogs suitable for study but there was no correlation between electrocardiographic changes and the appearance of the myocardium in the fifteen dogs so studied. One important point which may have clinical significance is that a group of dogs receiving 0.5 mg./kg. of cedilanid intravenously daily plus 1.5 mg. of atropine sulfate subcutaneously twice daily lived three times as long as dogs receiving the same doses of cedilanid and no atropine. The fact that in the former group (given atropine), sialorrhea, anorexia, and emesis were inhibited is cited as indicating that vagal inhibition prolonged life in dogs receiving cardiotoxic doses of cedilanid.

Rosenblum et al.[29] in a small group of dogs confirmed the work of LaDue,[11, 28] as to the production of myocardial necrosis from prolonged intravenous cedilanid, six out of eight dogs showing focal necrosis but in addition he found that each of two dogs given digitoxin intravenously had even more necrosis. This series is probably not conclusive because of the small number of animals involved.

SUMMARY

A critcal survey of the literature regarding many phases of experimental study on the cardiac glycosides with special reference to lanatoside C (cedilanid) has been presented, together with an attempt at fitting together the picture of the importance of these experimental findings as regards clinical medicine.

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CONGENITAL CYSTIC DISEASE OF THE LUNG

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SYNONYMS

Because of the variety of terms used in the past in discussions on congenital cystic disease of the lung, it is important to record them in order not to lose contact with the earlier records of this condition. I have found eighteen different terms in the literature that I have read and there are undoubtedly others. They are: (1) congenital cystic disease of the lung, (2) congenital cystic formation of the lung, (3) congenital cystic malformation of the lung, (4) atelectatic bronchiectasis, (5) congenital bronchiectasis, (6) honeycomb lung, (7) pulmonary cysts, (8) emphysematous bullae, (9) vesicular pulmonary emphysema, (10) bullous emphysema, (11) pleural blebs, (12) chronic interstitial pneumonitis with emphysema, (13) pulmonary pneumatoceles, (14) pneumatocele, (15) pneumocele, (16) pneumocyst, (17) air cyst, and (18) balloon cyst of the lung.

DEFINITION AND CLASSIFICATION

Congenital cystic disease of the lung is difficult to define because of its variety of types and manifestations. It might be defined as any congenital cystic condition connected with the respiratory tree. It may or may not be symptomatic, depending on the amount of lung tissue displaced and on the presence or absence of infection. It may be bilateral or unilateral. The cysts may be single or multiple and may or may not contain liquid.

I shall present two classifications; namely those of Koontz and Sellers.

Koontz's Classification

> "a. Bronchial dilations, as shown by the concentric muscle fibers in the walls, cartilage at times, and the lining of stratified ciliated columnar epithelium.
>
> "b. Cavities resembling emphysematous blebs lying subpleurally."

Sellers' Classification (modified from Cheney and Garland)

1. Solitary cysts—almost invariably of large size
   a. Huge "balloon" or "distention" cysts in infants causing gross pressure signs and often death.
   b. Smaller cysts about the size of an orange. These are often silent clinically unless they become infected.
2. Multiple cysts of varying size

a. Medium size, in groups of two or three close to the root of the lung. These are cherry to golf ball size, usually not clinically noted.

b. Small cysts, of currant to cherry size.

1. Following a set distribution usually lobar ni character. Conformity in size of cysts (honeycomb lung).
2. Diffuse or scattered lesions with cysts inconstant in size.

Neither of these classifications includes the congenital buds from the primitive foregut which are lined by gastric or intestinal mucosa.

HISTORY

The first mention of this condition was in 1638 in a report from Fontanus to Claudius Plempius. In 1687 Thomas Bartholinus reported three cases of congenital cystic disease of the lung and included this original case in the three. The patient was said to have died because the heart could not be properly cooled by the air from this vesicle.

Until 1925 the condition has been of academic interest only. In this year Koontz reviewed the literature and found 108 cases reported since the seventeenth century. None of these were in the American literature. In 1937 Schenck found 381 cases, an increase of 243 cases in twelve years. The wide use of the x-ray, especially in the Army and Navy and in routine examination for tuberculosis and the rapid advances in thoracic surgery, have caused universal interest in this condition. Reports are not at all uncommon now.

INCIDENCE

Congenital lung cysts are of equal incidence in both sexes. There have been reported cases in ages varying from premature infants to extreme old age. There is a difference of opinion as to the incidence. Pierce says that a true congenital cyst is a "rara avis." Greaves states that the increased incidence is due to the increased search for tuberculosis. No doubt there are numerous cases of cystic disease of the lung that are unrecognized when present in an x-ray. There are also many people whose cysts cause them no symptoms and therefore they are never diagnosed. It would be very difficult to determine the actual incidence. There has been a marked increase in incidence of reported cases since Koontz's paper in 1925. Brown and Brock state that the highest incidence of symptomatic cysts is in infants.

If those cases, wrongly diagnosed as types of bronchiectasis and as other conditions, were included the incidence would be much higher.

ETIOLOGY AND PATHOLOGY

The etiology of congenital cystic disease of the lung is not definitely known. This is shown by the great number of theories as to its cause. It is agreed by most authorities that the condition is congenital. The fact that when the condition is discovered in old age and the lung collapsed by the cyst is examined it does not show any pigmentation which lung tissue that has functioned shows. This proves that the lung was collapsed before birth.

I shall give a short statement as to the various causes as advanced by several different men who have given opinions as to the cause of the disease.

Grawitz believes that the condition is due to "hydroptic cystic malformation" due to faulty anlagen of the lymph system of the involved portions of the lung. The cysts, according to him, are of this origin.

Stoerk states that there are two types of cystic disease of the lung: (a) that due to fetal bronchiectasis secondary to infection with stenosis and closure of the bronchioles of the fetus. The cysts are said to be due to the collection of secretion distal to these obstructions; (b) that due to the excessive growth of the parts surrounding the bronchial tree, especially the connective tissue with retention of the fetal characteristics, that is, blunt, stubby tubes.

Sandoz puts forth the idea that it is due to faulty alveolar development from the small end bulbs of the bronchial tree in the embryo. He attributes this to congenital lues.

Kimla attributes the development of congenital lung cysts to excessive growth of the mesodermic structures with a parceling out of the glandular structures, which then undergo cystadenomatous change.

Couvelaire's idea is that endoderm is the essential factor necessary and sufficient for cyst formation.

Buchmann stated that the cysts were really tumor changes as shown by the strong development of the smooth muscle and epithelial proliferation.

Humbert, along with Balzer and Grandhomme, thought that syphilis played an important part in the development of lung cysts and that if the condition was not present at birth, it might develop later due to some congenital defect.

According to Heuter, the cysts develop because of a closing off or stenosis of the bronchial branches. He stated that the earlier the process develops, the fewer and larger the cysts. If the process develops late the cysts are more numerous and smaller. This idea is similar to that of Stoerk.

Heller said that it was a result of faulty expansion of lungs which were normal at birth.

Oudendal thought that the regular character of the cyst wall showed that it was not a neoplasm. He thought that fetal adhesions caused the cystic condition.

Box attributed the condition to bronchopneumonia.

Kanthuch and Rolleston described the cyst cavities as being broken down abscess cavities, but at the same time they described the walls as being smooth and glistening.

King and Harris think that the bronchi develop as small ramifications of entodermal tissue which become canalized. According to them this canalization occurs distal to some noncanalized part and the mucosa secretes mucous and causes the development of a cyst.

Most of the theories as to the cause of cystic disease of the lung are included in the above theories. Some are parts of more than one of the above.

As has already been stated in the classification, the cysts may be single or multiple, large or small and fluid containing or air containing.

Koontz found an incidence of 50 per cent in the left lung, 25 per cent in the right lung and 25 per cent in both lungs.

Schenck found 38 per cent solitary cysts, 62 per cent multiple and 23 per cent fluid containing cysts.

Cysts may or may not contain fluid. Those cysts which contain fluid may rupture into a bronchus. When this happens the cyst may collapse or it may be filled with air.

The wall of the cyst may have several types of lining. The lining may be greatly altered by infection. There may be no epithelial lining at all.

The cyst or cysts may be within the lung parenchyma and the structures of its wall are continuous with those of the bronchi and of the same tissue. There may be concentric smooth muscle fibers in the walls. At times cartilage is present and the cyst may be lined with ciliated stratified columnar epithelium. The cyst may be a subpleural bleb which closely resembles the subpleural bleb of emphysema. The cysts may be extraplumonary, arising apparently from rests or herniations of the bronchi or the trachea near the root of the lung.

The lining may vary. Any type of epithelial tissue may be present or as has been said before, none at all may be present. The arrangement of the elements of the cyst wall may be very irregular and the elements appear clumped together. Mucous glands may or may not be present.

Cysts may or may not be directly connected with the lumen of the bronchi. This is important. The type of connection is also important as this may be the determining factor in whether or not the cyst is symptomatic.

If a cyst contains fluid and ruptures into a bronchus it may become an air containing cyst. If a ball valve mechanism is set up so that air goes into the cyst but cannot escape, the cyst may be markedly increased in size. This is the balloon type cyst. They are usually, but not necessarily, single. The valve mechanism may not be constant so that the size may vary markedly from day to day. This is the type cyst that may cause a sudden onset of symptoms. The smaller cysts and the fluid containing cysts usually remain unchanged unless infection is present in them. Those air cysts that are not expansile usually have a free connection with a bronchus.

Womack and Graham found evidence of a spindle cell invasion into a large number of pulmonary cysts. The lesions resembled bronchiogenic carcinoma closely, but they found no evidence of metastasis.

It is not uncommon for the larger cysts to have strands of lung tissue or bronchi passing through them. These show up in the roentgenogram as fine trabeculations traversing the cyst cavity.

Ward and Krahl described a case of cystic disease of the lung in which the cysts were lined with gastric mucosa. The cysts were not in any way connected with the gastrointestinal tract. One of the cysts communicated with a bronchus. They suggest that it was due to a pinching off of a part of the foregut in the third or fourth fetal week.

Congenital cystic disease of the lung is frequently associated with other congenital anomalies.

SYMPTOMS AND SIGNS

Most congenital lung cysts are asymptomatic. They are commonly diagnosed by some by routine x-ray or at autopsy where there has been no respiratory difficulty. The symptoms and signs associated with this condition are usually due to infection or to encroachment on the functional lung tissue by the cysts.

Cheney and Garland described a case of a 19 year old girl with a large cyst in the left lung which lowered her vital capacity to 1200 c.c. (27 per cent of normal). She danced, swam, and played tennis with no apparent discomfort. Others, however, have described cases with much less involvement in which the signs of respiratory embarrassment were very evident. This is especially true in cases in which an apparently silent cyst becomes markedly and rapidly enlarged due to a ball valve mechanism and suddenly encroaches on the surrounding lung. Such attacks may be intermittent due to changes in the type of connection with a bronchus.

As a rule, the small honeycomb cysts do not give rise to difficulty in the adult unless they are infected because there is no great or rapid change in their size. They are more likely to cause respiratory difficulty in infancy because they replace normal functional lung. If they are not extensive enough to cause decreased lung function in infancy they usually cause no distress in the adult.

There is no pathognomonic sign of cystic disease of the lung. The most commonly found signs are cough, dyspnea, wheezing, expectoration and hemoptysis. In the ball valve type of balloon cyst there may be a sudden onset of respiratory distress with cyanosis and death due to suffocation. At birth respiratory distress may be due to a lung cyst.

There may be some expectoration due to a non-infected cyst. It may be increased by changes in position.

When a cyst becomes infected fever and expectoration are usually prevalent along with the other symptoms mentioned.

In addition to the respiratory symptoms there may be marked anorexia and loss of weight, thoracic pain, vomiting and palpitation.

Physical examination findings vary with the type of cyst and the number and location of the cysts. Small cysts deep in the lung may show no signs whatever. A large tension cyst may cause a lagging of the chest on the affected side and may give a tympanitic note to percussion. The breath sounds are usually absent unless air is coming into the cyst. Fluid containing cysts, which are large, give the physical signs of any solid mass.

Kaltreider and Fray observed six patients with lung cysts. They found that in three of the cases the vital capacity and total capacity were slightly reduced and the residual air was slightly increased. The volume of the chest was increased in all cases, that is, the antero-posterior diameter was increased. There was slight carbon dioxide retention in two and slight anoxemia in four. They found dyspnea to parallel the decrease in vital capacity.

X-ray is the most valuable diagnostic aid. Fluid containing cysts show up as areas of increased density. The air containing cysts show up as areas of decreased density with fine trabeculations. By injection of lipiodol and having the patient shift position the cavity of a cyst communicating with a bronchus may be outlined. Intracystic pressure reading may give some idea as to whether there is a patent connection with a bronchus. X-ray is not absolutely diagnostic. Exploratory thoracotomy may be the only absolute way to make the diagnosis.

DIFFERENTIAL DIAGNOSIS

Infection in a lung cyst may be mistaken for empyema. Such an error may result in prolonged treatment with poor results. Empyema with a bronchopleural fistula may even more nearly simulate an infected cyst. With empyema there is usually retraction of the chest on the affected side and there may be an approximation of the ribs. At operation the presence of a periosteal reaction with thickened ribs suggests empyema. X-ray may be of some aid. Cysts are usually spherical or oval whereas empyema is usually triangular or round and conforms to the contour of surrounding structures. There is little or no pleural thickening in cases of lung cyst. This is an important differentiation because an epithelial lined cyst will not close even though adequate drainage is effected. Biopsy of the cavity wall will determine this point.

Pneumothorax is another condition commonly mistaken for cystic disease of the lung. This is especially true of the large tension cysts. X-ray of pneumothorax shows collapse toward the hilus. This picture is altered by the presence of adhesions. As has been said before there are frequently fine trabeculations seen in cysts due to the bands of lung tissue or bronchi traversing their lumens. Cyst pressure is exerted in all directions and some lung density may be seen on all sides of the area of rarefaction. Pressure readings within the cyst or pneumothorax may be helpful but at times this procedure may be dangerous.

Herniation of the stomach or bowel into the thorax may give rise to a picture similar to cystic disease. Barium meal will rule this condition out. A large gas bubble in the stomach, especially with an elevation of the left diaphragm may be mistaken for a cystic lung at first glance.

Infected cysts may very closely resemble a lung abscess. It may be impossible to make a differential diagnosis. There may be a fluid level in both and a surrounding area of pneumonitis. Usually the cyst does not cause as much reaction, both local and general because of its epithelial lining. At times there is an epithelialization of an abscess cavity and a definite diagnosis cannot be made by biopsy. Lung abscess frequently has a history of operation in the nose or throat or aspiration of a foreign body.

At times this condition may be confused with tuberculosis. There is no positive sputum, constitutional symptoms are less, the Mantoux may not be positive in the former. Serial x-rays may be necessary to form a definite diagnosis.

Bronchiectasis may closely resemble cystic disease of the lung. Bronchiectasis is usually in the lower lobes. Schenck said many cases diagnosed bronchiectasis may very likely be in reality lung cysts. There is an overlapping of some types of each. It is frequently impossible to make a definite diagnosis when infected cysts are present.

Fluid containing cysts may resemble a dermoid cyst, echinococcus cyst, teratoma, chondroma, ganglioneuroma, and other thoracic tumors. Definite diagnosis can only be made by exploration.

Emphysema may show up in an x-ray as areas of decreased density similar to air cysts. Usually some causative factor of emphysema can be demonstrated.

TREATMENT

No set method of treatment can be given for cystic disease of the lung. The number, distribution, and size of the cysts are too inconstant to follow set rules.

Most cysts are asymptomatic and require

no treatment. In those cysts with a ball valve mechanism in which there is a rapid increase in size immediate treatment is necessary. Aspiration usually affords temporary relief. Pneumothorax is of no value because it collapses what lung is left and does not collapse the cyst. Also a pressure pneumothorax may develop. Brown and Brock used a continuous suction of the Monaldi type which collapsed the cyst and re-expanded the lung. They then made an endocutaneous flap into the cyst and connect it to the outside.

Sclerosing the bronchial opening and surgical closure of these cysts has not been very successful. Frequently it results in further stenosis without complete closure making the condition worse.

Enucleation is usually not feasible because the wall of the cyst is continuous with that of the surrounding lung.

Miller and Lillienthal inserted a one way valve through the chest wall into a cyst and got a collapse.

Collapse of a cyst will not result in permanent closure because the cysts are usually lined with epithelium.

Cauterization of the cyst wall may or may not result in closure.

Lobectomy or pneumonectomy have given the best results in the treatment of lung cysts, especially those of honeycomb type limited to one lobe or one lung and the large tension cysts.

Those cases in which the cysts are widely spread and in which operation is contraindicated must be treated conservatively. It is sometimes helpful to have such a patient move to a dry, warm climate to decrease the incidence of respiratory infection. The instillation of lipiodol has been helpful in clearing up some infected cysts.

PROGNOSIS AND COMPLICATIONS

The prognosis in congenital cysts of the lung is generally good. Most cysts are discovered accidentally or at autopsy where the patient died of some other disease.

Infection, especially in fluid containing cysts may be quite serious, especially if the extent of the disease contraindicates surgery.

Infection of a cyst is probably its most serious complication. Kirklin found tuberculous infection to be less than 5 per cent.

A sudden increase in size in a cyst may cause sudden death if treatment is not immediately instituted.

Spontaneous tension pneumothorax may result from rupture of a cyst into the pleural cavity, especially where aspiration of the cyst is attempted.

Cor pulmonale and heart failure may develop due to the increased pulmonary pressure.

SUMMARY

Congenital cystic disease of the lung is a condition which is not rare. It is very frequently asymptomatic and found at routine examination or as a casual finding at autopsy. The etiology is obscure, but most opinions are centered around faulty development of the bronchial buds. It may be confused with empyema, spontaneous pneumothorax, tuberculosis and numerous thoracic tumors. Frequently it cannot be distinguished from some cases of bronchiectasis even by the pathologist. Infection and sudden enlargement are the two most dangerous complications of the condition. There is no routine way of treating. Each case demands treatment according to its type. Inoperable cases may be helped by moving to a dry climate to reduce the incidence of respiratory infections.

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THE BONE MARROW IN DISEASES OF THE LEUKOPOIETIC SYSTEM

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HISTORY

Until seventy years ago bone marrow was an unexplored tissue. It is only within the last 35 years that the bone marrow has been employed during life as a means of diagnosis. Ghedini, in 1908, reported the first study on the subject when he biopsied the tibia and demonstrated the presence of parasites in cases of malaria and leishmaniasis. Seyfaith, in 1923, improved on this by selecting the sternum as the site of biopsy, because the sternal marrow was more active throughout life and was more accessible. Then, in 1927, Arinkin introduced the aspiration method which has been very widely used and added greatly to the knowledge of the cytology of bone marrow!

PURPOSE OF PAPER

It is the purpose of this paper: (1) to familiarize the reader with the origin and morphology of the cells of the normal bone marrow; (2) to give in a detailed manner the technic of sternal puncture and in a generalized manner other less widely used methods of bone marrow study; (3) to give the clinical indications for sternal biopsy; (4) to inform the reader what conditions produce marrow changes which are specific; and (5) to aid the reader in establishing a diagnosis of the more important leukopoietic diseases by bone marrow examination.

For reasons to be presented later the biopsied material here described was obtained by the sternal puncture method.

NORMAL MYELOGRAM

In order for one intelligently to study the bone marrow in pathologic states, a working knowledge of the normal marrow is practically indispensable for proper interpretation. Schilling coined the name haemogram to designate the qualitative blood picture; likewise the term myelogram can reasonably be applied to the marrow picture.

Origin of Cells: There are two trains of thought concerning the origin of blood cells during postnatal life. One group (the monophyletic school) contends that the cells of the blood are derived from a single primitive stem cell, whereas the other group (the polyphyletic school) contends that the various blood cells have individual precursors and separate cycles of maturation.

Notable figures such as Pappenheim, Maximow, and Downey are of the monophyletic school and adhere to the idea that a single polyvalent cell, the "lymphoidocyte" or hematocytoblast is present in all adult hematopoietic tissues and this cell gives rise to the other cells of the blood.

The polyphyletists, on the other hand, deny any such cell as the hematocytoblast but differ among themselves concerning the number of leukoblastic stem cells: the dualists contending that there are two stem cells, lymphoblast and myeloblast; and the trialist contending there are three stem cells, lymphoblast, myeloblast and monoblast. The latter is the most widely accepted theory. It not only postulates an individual stem cell for every type of blood cell, but advocates a different site of origin in normal postnatal life.

Cytology: As has been mentioned, it is absolutely essential that one have a working knowledge of the cells in the normal marrow in order to recognize the marrow in pathologic states. It is evident that there is still much diversity of opinion as to the stem cells of the various cell series. There are also considerable variations as to the appearance of the normal marrow. This has led some workers (Propp and Schwind[4]) arbitrarily to grade the cellularity of the marrows in their series into four groups or grades. Grade 1 marrows have approximately the number of nucleated cells found in peripheral blood smears. Grade 2 marrows have four to five times this number of cells, and Grade 3 marrows have from ten to twenty times this number. In Grade 4 marrows there are more nucleated cells than erythrocytes, and the cells appear on the smears in clumps and masses. According to these workers, Grade 2 is the most common. Grade 4 type occurs only in abnormal conditions where the marrow is extremely hyperplastic.

In studying marrow films, several points should be kept in mind.[5] Marrow films, unlike blood, have a semi-solid matrix background in which lie parts of cells, naked nuclei and cell granules. In determining the percentages of the various cell types in marrow films, a larger number of cells should be counted than is customary in blood counts, because there are usually more cells and they are far from being evenly distributed. Piney[5] says that at least one thousand cells should be examined, preferable in several films. The relative proportions of red and white cells must be determined as in the blood, but in comparing them with the differential count of the peripheral blood separate myelograms of the white cells should be obtained.

In order to avoid any further confusion regarding the cytology of bone marrow, I have selected Piney's description of the various cellular elements of the normal marrow.[5]

Since all cells, regardless of type, are thought to originate from some stem cell belonging to the reticulo-endothelial system, the haemohistioblast (one of its members) has been described by Ferrata as being the stem cell. It is so primitive and undifferentiated that it is capable of developing into connective tissue or into blood. These cells are about fifteen to twenty microns in diameter with a delicate, irregular, slightly basophilic cytoplasm. The mucleus is round or oval, of fine, delicately reticulated structure in which one or more distinctive nucleoli are found. Recognition in marrow films is by the nuclei, since the cytoplasm is so delicate that it usually ruptures.

The nature of the hematocytoblast is still not definitely determined. Suffice it to say that it is smaller in size and more definite in shape than the more primitive haemohistioblast. The cytoplasm is devoid of granules, distinctly basophilic, and varies considerably in amount. The nucleus is denser than that of the haemohistioblast having interlacing fibrils of basi-chromatin in which one or more nucleoli are present.

The myeloblast, although very difficult to distinguish from the hematocytoblast, has a nucleus which is less dense and cytoplasm which is less basophilic. In any case, definite differentiation is practically impossible.

The premyelocyte is an intermediate cell type between the myeloblast and the myelocyte, and there is no doubt as to its myeloid nature. The nucleus is denser and more deeply stained than that of the myeloblast, but nucleoli can still be detected in it. The cytoplasm is homogenous and less basophilic than that of the myeloblast.

Two grades of premyelocytes are described: those having immature, azurophilic granules; and those having granules of the same tinctorial properties as mature cells. The former have nucleoli resembling those of myeloblasts, whereas the latter have a denser structure.

As with the more mature forms, three varieties of immature premyelocytes are described: those with minute azurophilic granules, all the same size—preneutrophilic premyelocytes; those with larger rather scanty azurophilic granules, all the same size—pre-eosinophilic premyelocytes; and those with large irregular azurophilic granules, of different sizes—pre-basophilic premyelocytes. These, by maturation, give rise to premyelocytes with their respective types of granulation; and these in turn give rise to myelocytes.

Myelocytes vary greatly in size (10-25 microns). The nucleus is circular, but the edge may appear to be indented by the granules that fill the cytoplasm. The basichromatin has still a detectable reticular arrangement, but condensations may also be seen. The cytoplasm is fairly basophilic, but almost obscured by granules which have attained their definite character—neutrophilic, eosinophilic, or basophilic.

Metamyelocytes represent an intermediate stage between myelocytes and mature polymorphonuclears. Schilling has divided these into two groups: juvenile forms which have a slight indentation of the nucleus; and staff forms with deeply indented, but not lobulated, nuclei. The nuclei of the eosinophiles are more difficult to recognize, because they are pale and covered by large granules. The nuclei of the basophiles are even less distinct, and frequently it is impossible to recognize the two types of metamyelocytes.

The polymorphonuclears as seen in marraw films are identical in character with those found in the peripheral blood; and a description of their structure is not deemed necessary here.

Lymphocytes, although in a sense not constituents of the normal marrow, are found in small numbers. They occur only in groups, sometimes even with a germ-center. There is some evidence that suggests that the marrow may, in a small way, be a site for the production of lymphocytes since the marrow contains more large lymphocytes than does the blood.

The small lymphocytes of the marrow resemble those found in the peripheral blood; and the large forms differ from the small forms only in the amount of cytoplasm they contain; the nuclear characters are the same..

During health few, if any, immature cells of the lymphocytic series are found in the marrow. The lymphoblast is the early cell form from which the lymphocytes arise; and it is irreversibly determined in the direction of lymphocytopoiesis. Its structure is about the same as the myeloblast, but the nucleus is relatively larger and occupies most of the cell. It is reticulated rather more coarsely than that of the myeloblast, and contains nucleoli, around which the basi-chromatin is slightly condensed.

Monocytes are scanty in normal marrow and are identical with those found in the peripheral blood.

Plasma cells, although scanty, are normal constituents of the marrow. The cells are large (15-20 microns), have an eccentric nucleus with the basi-chromatin arranged in a radial pattern. The cytoplasm is intensely basophilic throughout, except for a peri-nuclear pallor.

Turk cells are very similar to plasma cells except the nucleus is not eccentric. In fact, some workers contend that they are the same cell—the turk cell being an immature form of the plasma cell. These, like plasma cells, are also scanty in the normal marrow.

Megakaryocytes are fairly numerous and important constituents of the normal marrow. These vary from 30-90 microns in diameter; and have a very faintly basophilic cytoplasm in which groups of minute azurophilic granules are present, especially near the periphery. The nuclei are whorled and lobulated in a most complicated manner; basi-chromatin occurs in large masses without any definite distribution. Complete cells are rarely found in marrow films, and the recognition of the fragments present is very difficult.

Since the scope of this paper is confined to the leukopoietic system, the appearances of the various hemoglobiniferous elements need not be described. Suffice it to say that the normoblastic series begins with the hematocytoblastic stage, as do other blood cells, and ends with the development of the ordinary red corpuscles.

As the normal marrow varies in other respects, so do the percentages of the different cells vary. This may be actual or may be due to inevitable errors. According to Piney, the following table gives the normal variations.

Neutrophiles:	Per cent
Myelocytes	30-35
Metamyelocytes	10-15
Polymorphs	24-30
Eosinophiles	
Myelocytes	1-2
Polymorphs	½-1
Basophiles:	
Myelocytes	3-6
Polymorphs	1-2
Premyelocytes	1-2
Hematocytoblasts, including myeloblasts	1-2
Haemohistioblasts	¼-½
Lymphocytes:	
Small	6-9
Large	8-12
Plasma cells	½-1
Pro-erythroblasts	1-2
Normoblasts	15-20
Megaloblasts	2-3
Megakaryocytes	scanty, but invariable

BONE MARROW BIOPSY

1. Trephine methods:[6] This method was first introduced by Pianese (in 1903) who obtained marrow for examination by trephining the femur. At present, however, the sternum is the site of choice. Even though this is an operating room procedure and is not the most widely used method, it is considered by Custer[6] and other workers to be the method of choice.

The procedure is essentially that of obtaining a button of bone with a 1 cm. diameter trephine from the ventral surface of the sternum, fixing it in Helly's fluid, decalcifying, embedding the specimen in paraffin, and then sectioning the specimen from its deep surface. The sections should then be stained with Maximow's Azure II eosin method. Fragments of marrow may be curetted from the margin of the cavity in the bone, pressed against, and drawn across clean micro slides and stained. Tissue cultures and supravital preparations may be made when indicated.

It must be remembered that dry imprints should be examined along with sections, because intrinsic cellular detail is clearer, blood smear comparisons are possible, and a correlation between the peripheral blood picture and marrow picture can be obtained.

2. *Aspiration method:*[4] This method of bone marrow examination is more widely used than any other because of its simplicity, but the novice should be forewarned of the technic, because his first attempts are apt to be unsatisfactory.

This technic consists in using 18 gauge spinal type needles with a heavy finger tip and stylet. Needles are cut in 2, 1.5 and 1 cm. lengths, and are used depending upon the size of the individual. Puncture is made in the midline of the body of the sternum at the level of the third costosternal synchrondrosis. Other levels may be used, if this site is not available, or if a check is desired. Probably in children, it is better to make the puncture at the level of the third interspace, because not infrequently the first and second sternebrae dot not unite until adult life is reached. One must maintain strict surgical asepsis. The skin, subcutaneous tissue, and periosteum are infiltrated with a total of about 3 c.c. of novacaine. This will usually produce the desired amount of anesthesia. The puncture needle is inserted vertically, and, finally, the outer table of the sternum is pierced by a rotary movement of the needle under firm pressure. Boring is continued until the needle becomes fixed in the bone. The stylet is removed and a tuberculin syringe or large syringe, if necessary, is attached and suction exerted by pulling on the plunger. If no marrow appears, the needle is advanced farther and aspiration is tried again. This is continued until the appearance of gross blood at the distal end of the syringe. This should be about 0.2 c.c. Suction should then be stopped, and the needle and syringe withdrawn. Care should be taken to hold the syringe with its attached needle horizontally to prevent extrusion of drops of marrow from the end of the needle.

Very little pain is produced in performing a sternal puncture. Occasionally, however, some pain is produced when the periosteum is pierced by the infiltrating needle, and one may experience a not severe aching in the chest. This is transitory however, and is probably due to the temporary negative pressure in the marrow cavity. After the needle is withdrawn, a sterile compress is applied to the puncture wound and held in place with adhesive for 48 hours.

It has been found that the most satisfactory portion for study is that which enters the needle last, and, therefore is used for making the first smears. Anticoagulants are to be avoided, because they interfere with staining. Therefore, one must work rapidly since clotting occurs quickly in the needle. The needle and syringe are held horizontally with the opening of the needle face down, and small drops of marrow are made to appear at the opening by pressure on the plunger. The drops should be small, and at least twelve dried smears are made on clean cover slips. The cells on the smears should lie close together but not overlap. Smears that are too thick do not stain well; if too thin, the more fragile cells will break up. If smears are not dried quickly, they will not stain properly. Either Wright's stain or the May-Gruenwald-Giemsa stain may be used. The former is by far the easier and more widely used of these stains.

Reticulocyte counts, for the sake of uniformity, are made from the second drop of marrow coming from the needle. The material is smeared on a slide and stained according to the brilliant cresyl blue technic.

Although supravital preparations are made with every puncture by some workers, they are not an essential part of the technic but may be of value in the identification of monocytes.

3. *Comparative value of methods presented:*[6, 7] Even though bone marrow studies of the hematopoietic system are relatively recent, their value cannot be overestimated. By employing the trephine method, one can determine the state of hyperactivity, hypoplasia or defective maturation in one or all of the cell series; whereas in aspirated marrow these matters are questionable, because the cells do not retain their original relationship, probably not their relative proportions, and the material withdrawn is apt not to be representative. Focal lesions of diagnostic value are very apt to be missed, and aspirative marrow is often unavoidably diluted with blood. Lastly, the cells are subjected to more trauma and are more likely to be damaged in preparing marrow-blood mixtures, whereas in sections they are well preserved.

The aspiration method is more widely used because of its simplicity, and repeated biopsies on the same individual may be performed. It is also quite satisfactory in certain instances, since it enables one to make a diagnosis without employing the more radical procedure of trephining. One should employ both procedures if necessary, using the trephine method only when the more conservative aspiration method fails to give the desired information.

4. *Drill method:*[6] Favorite compromised between the methods mentioned above and devised a drill method. This consisted of a drill ensheathed by a cannula. After introducing the drill into the marrow cavity, it is rotated and fragments are withdrawn from which imprints and sections can be made. By retaining the cannula in place, one can introduce the drill at different angles thereby obtaining more material and examining a larger area.

CLINICAL INDICATIONS FOR BIOPSY

The question arises, when is the sternal puncture method of marrow biopsy indicated? According to Propp and Schwind,[4] it should always be performed when: (1)

the diagnosis is in doubt, and a disease usually showing a typical myelogram cannot be excluded; (2) the etiology of the peripheral blood is obscure; (3) other means of biopsy have failed. It may be done: (1) to save time, if a specific marrow picture is likely to be present; (2) to obtain information concerning prognosis; (3) to determine response to treatment; (4) to determine the type of "stem cell" leukemia when this cannot be done from the peripheral blood. It should also be performed whenever splenomegaly is present.

MYELOGRAM CHANGES IN LEUKOPOIETIC DISEASES

A. *Specific and non-specific myelograms:*[4] Sternal puncture findings fall into two categories, specific and non-specific. Myelograms which are typical of a particular disease, and which when present are so reliable as either to indicate the diagnosis or to be actually pathognomonic, are termed specific or typical myelograms. These are found in the following conditions: (1) leukemia, especially acute and leukopenic leukemia; (2) megaloblastic marrow, present typically in pernicious anemia in relapse, achrestic anemia, and sprue; (3) agranulocytosis in the arrested marrow stage; (4) neoplasms: multiple myeloma and metastatic malignancies, especially carcinoma; (5) lipoid histiocytosis; (6) malaria, and (7) leishmaniasis (kala-azar).

It should be remembered that the above conditions will not always give diagnostic marrow pictures, but when specific findings are present, they do indicate the diagnosis. Bizarre cases may be encountered which confuse the picture very much. The absence of a typical myelogram cannot be relied upon to rule out a disease in differential diagnosis, because of the possibility of an error in sampling or atypical variations in pathology. But negative findings may be extremely useful in some instances.

All other diseases so far studied constitute the non-specific group. Although the myelograms in this group are non-typical, they are often of great interest and usefulness in relation to the general clinical picture.

B. *Pathological states:*

1. Leukemias: Even though the bone marrow picture in the various forms of leukemia are rather complex, they are more clearly defined than any other disease entity affecting the hematopoietic system. Frequently it is possible to make a diagnosis clinically employing the ordinary blood examination to confirm it, discover the type and give some indication of the severity of the disease. But the marrow examination reveals a much clearer insight to the severity, changes indicative of approaching relapse and valuable information concerning therapeutic control.

a. Chronic myeloid leukemia:[5] Marrow examination is not of much value in the early stages of this condition, because slight changes in the marrow are difficult to recognize, as has been mentioned.

During health, mature polymorphs are thought to be formed by division and maturation of myelocytes and development from the less mature types, such as the myeloblast plays little, if any, role in granulopoiesis after birth. Likewise, the normal mechanism persists to a considerable degree in chronic myeloid leukemia.

When definite changes in the bone marrow have developed, one finds a marked increase in the cellularity. This increase consists mainly of myelocytes which may, in some cases, constitute as much as 80 per cent of the white cells present. Most of the white cells are neutrophiles, but eosinophiles and basophiles are also increased. Some myelocytes contain both eosinophilic and basophilic granules. The cells are larger and less mature than the corresponding cells found in peripheral blood. The following table shows a fairly typical myelogram in a case of chronic myeloid leukemia.

Total Nucleated cells 70,000 but variable

	Per cent
Myeloblasts	1.1
Promyelocytes	0
Myelocytes	37.4
Nonsegmented neutrophiles	31.0
Segmented neutrophiles	20.0
Eosinophilic myelocytes	5.6
Nonsegmented eosinophiles	0

Eosinophiles	3.4
Lymphocytes	0
Monocytes	0
Megaloblasts	2.0
Normoblasts	2.2
Megakaryocytes	0.2

Two other important but difficult points to determine are: (1) the background of the film (matrix) in chronic myeloid leukemia is denser and contains more granules and debris than is found in normal healthy marrow, and (2) the cells (or some of them) tend to lie in groups which are all composed of a single cell type. Mitotic figures are most frequently seen in these cell groups, and if such is the case with myeloblasts, it is suggested that the morbid process is becoming more active.

Patients with leukemia should have sternal punctures performed periodically, and when there is a great increase in the number of cells less differentiated than myelocytes, it is indicative of an eminent relapse, of an acute exacerbation. One can thus be forewarned of such changes, since changes in the bone marrow precede the changes in the peripheral blood.

It is important to examine the marrow very carefully in the early stages, since it is extremely easy to mistake the appearance of the marrow for that of a simple reactive hyperplasia, such as may be associated with any infection, because in chronic myeloid leukemia there are, in addition to the leukoblastic changes, erythroblastic and megakaryocytic reactions.

b. Chronic lymphatic leukemia:[5] In this condition the myelogram is usually easy to interpret, since lymphocytes are not numerous in the normal marrow, and never show signs of activity. The lymphocytes are very closely packed together, but may be interrupted in various places by small nests of red cells and granulocytes. The background of the film is moderately thick, but hyaline, and unlike that seen in chronic myeloid leukemia.

Small lymphocytes, with a variable proportion of large forms, constitute most of the cells. Cells of the lymphoblastic group are usually scarce. An increase in cells of this series implies active progress of the disease. If lymphoblasts are very numerous, the prognosis is grave, and in all probability radiotherapy will only aggravate the condition. In early cases, if the focal arrangement of lymphocytes is present, it is pathognomonic of the condition.

Although sternal puncture is rarely needed to diagnose chronic myeloid leukemia, it may be of utmost value in the diagnosis of chronic lymphatic leukemia, because atypical forms are quite common. This is especially true of chronic aleukemic leukemia which may be without any of the classical signs of leukemia (see aleukemic leukemia).

The myelogram in a typical fairly advanced case of chronic lymphatic leukemia is shown in table below.

	Per cent
Neutrophiles:	
Myelocytes	1-2
Metamyelocytes	1-2
Polymorphs	1-2
Eosinophiles	0-8
Lymphocytes:	
Small	30-40
Large	30-40
Immature	10-20
Blasts	5-15
Monocytes	2-5
Normoblasts	2-5
Megakaryocytes	2-4

c. Monocytic leukemia:[5] The exact nature of this condition is still uncertain, probably because it is a relatively uncommon type of leukemia. Most of these cases tend to be acute or subacute rather than chronic. Aside from this characteristic there is no clinical difference between monocytic leukemia and the other types of leukemia.

There is in this condition a massive proliferation of the cells of the reticuloendothelial system. This takes place to such an extent in the bone marrow, that the normal myeloid tissue becomes ultimately excluded. There is both a relative and absolute increase in the monocytic type of cells. These may form as much as 95 per cent of the total leukocytes. Most of the cells are of the classical kind, large, but varying in size. Younger forms may be found but are usually present in smaller numbers.

The fact that the immature elements are more numerous than fully differentiated ones, conforms with the knowledge that this condition is generally more rapid in its evolution.

In this condition the peripheral blood shows an increase in the number of white cells up to 400,000 per cu. mm. But here again aleukemia cases occur, and unless myelograms are studied, the diagnosis may be missed (see aleukemic leukemia). Although monocytes make up most of the cells ordinarily, the terminal stages are manifested by a marked increase in the monoblasts. Again, as was mentioned before, the changes in the bone marrow precede those in the peripheral blood, and for obvious reasons marrow examinations should be performed.

d. Acute leukemia:[5] This condition may be primary or it may be the terminal phase of a chronic case. The myelograms in these conditions are varied and might lead one to suppose that there is an infinite number of types of acute leukemia. Although in some cases the acute forms may be differentiated into myeloid, lymphatic and monocytic forms, there are many instances where the cells infiltrating the marrow are so undifferentiated and primitive that it is practically impossible to ascertain their nature. Such a condition can simply be termed acute leukoblastosis or acute leukemia. Piney has said, "all cases of acute leukemia are hematologically and, sometimes, clinically atypical." Other workers, on the other hand, say that in their series the marrow was invariably typical. These workers found that the normal cells were almost entirely replaced by lymphoblasts and lymphocytes in the acute lymphatic type, and by myeloblasts and myelocytes in the acute myeloid type. In all their cases of acute involvement, there was a marked diminution in the erythroid and megakaryocytic elements.

The monocytic variety may be differentiated from other types of acute leukemia by employing the peroxidase staining technic in much the same way that is employed in the peripheral blood examination.

The acute type of monocytic differs from the chronic type only in that the cells retain their fixed position in relation to the reticular fibrillae, whereas, in the chronic they become rounded up and free, often phagocytosing erythrocytes and debris.

e. Plasma cell leukemia:[6] In this condition, there is marked dissemination of plasmoblasts and plasmocytes throughout the myeloid cavities. These are more readily recognized from the characteristic morphology of the more mature forms. This can be differentiated from plasma cell myeloma by its diffuse type of proliferation and the absence of osteopysis.

f. Megakaryocytic leukemia:[6] There is much dispute that this condition exists as a separate entity. Little more needs to be said except that many workers have found it almost impossible to differentiate this condition from cases of chronic myelosis in which there is extensive megakaryocytic proliferation.

g. Aleukemic leukemia:[8] Diagnosis in this condition may be impossible, since the total leukocyte count of the peripheral blood may be normal or subnormal. Occasionally, however, a few immature leukocytes may be found in the peripheral blood but nothing diagnostic like one finds in the bone marrow. It has been estimated that 10 per cent or more of all leukemic cases are of this type.

It is the belief of most workers that this condition is just a stage in the leukemic process. Both acute and chronic forms occur, and all of the various types, myelogenous, lymphogenous and monocytic, have been described.

The clinical picture is essentially the same as that seen in the more typical forms of the various leukemias, except for the peripheral blood picture changes. The diagnosis depends upon the bone marrow picture which will show the typical myelogram changes which were discussed under the ordinary types of leukemia; of course, the findings will depend upon the type of leukemia present. Obviously, in these conditions it is very important that the marrow pictures be compared with the peripheral blood pictures.

2. Leukemoid reactions: Such reactions are of value here, because they are characterized by blood changes which resemble those of leukemia and must be ruled out in the differential diagnosis. The diagnosis based upon blood and marrow examinations is difficult because of the varied reacting power of the bone marrow; but even the possible types of reactions are limited. The difficulty in differentiation from leukemia is dependent upon whether the marrow reaction affects the erythrocytopoietic tissue mainly; in which instance the problem is less difficult than when the leukoblastic cells are involved, where differentiation is almost impossible. According to Piney, blood examination is more important for diagnosis than bone marrow examination in leukemoid reactions.

Krumbhaar[9] divided leukemoid reactions into two groups: (1) those presenting signs and symptoms suggestive of leukemia, and (2) those that resemble leukemia only from the hematological standpoint. Many and varied causes may produce leukemoid reactions, and it has been stated that all cases of supposedly leukemias which have recovered were none other than another type of leukemoid reaction. The bone marrow picture is, generally speaking, similar to that present in the peripheral blood. Hill and Duncan[10] have drawn up the following table which is very helpful in the laboratory differential diagnosis between true leukemias and leukemoid reactions:

True leukemias	Leukemoid reactions
1. Leukocytes are atypical, particularly the immature ones.	1. Immature as well as mature leukocytes show normal morphology.
2. Myeloblasts may be numerous, as high as 99 per cent.	2. Myeloblasts usually less than 10 per cent.
3. Immature erythrocytes are rarely increased in proportion to the increase of immature leukocytes.	3. Erythroblasts often increase in proportion to immature leukocytes.
4. Platelets are decreased often severely; may be increased only in chronic myelocytic leukemia.	4. Platelets are usually normal or increased but may be moderately decreased.
5. Progressive anemia which may become worse.	5. Anemia is variable depending upon the cause.

3. Essential thrombocytopenic purpura:[11] Mature megakaryocytes are formed from myeloblasts through a process called homoplastic hemopoiesis. Thus any disturbance which involves the stem cell (myeloblast) will ultimately interfere with the normal development of the megakaryocyte, thereby resulting in a qualitative and quantitative change in them. Since all this takes place in the bone marrow, examination of it represents the most important single differential diagnostic features.

In acute thrombocytopenic purpura, the myelogram reveals a uniform stimulation with a myeloid, erythroid, and most important of all, a megakaryocytic hyperplasia. In the more chronic stages, without hemorrhages, the myeloid and erythroid hyperplasia may be absent, but the megakaryocytic hyperplasia persists. In the acute phase of the disease, many of the megakaryocytes are young forms, whereas, in the more chronic phases, the megakaryocytes are adult in type and possess an abundance of azurophilic granules in their cytoplasm. Some of the megakaryocytes reveal hyaline changes in their cytoplasm, and the azurophilic granules may be absent. Degenerated or toxic forms with vacuolization of their cytoplasm are occasionally seen, but are more frequently seen in larger numbers in some cases of symptomatic purpura hemorrhagica, especially those due to infections and intoxications.

4. Agranulocytosis:[12] There has been much discussion concerning the etiology of agranulocytosis. In all cases where the etiology is in question, sternal marrow studies should be performed, because they may be of value in revealing the underlying cause, such as, leukosis or panmyelophthisis.

The bone marrow picture in this condition shows marked variations. These may be grouped into three types: (1) aplastic, (2) normal or hyperplastic, and (3) reticulo-endothelial.

a. Aplastic: The aplasia involves the granulocytes and myeloblasts. The megakaryocytes and erythrocytes are usually very little affected. The marrow picture shows infiltration throughout with lymphocytes and plasma cells. The aplasia may be partial or there may be complete absence of polymorphonuclear cells. When there is marked leukopenia with the complete absence of polymorphonuclear cells the prognosis is grave.

b. Normal or hyperplastic: In this type the marrow picture shows many myeloblasts and a few myelocytes, although the granulocytes are completely absent from the peripheral blood. Normal or hyperplastic marrows are often found in cases during remissions and in cases which have recovered. The finding of such a marrow picture therefore is usually indicative of a favorable prognosis.

c. Reticulo-endothelial: In this type, only a few myeloblasts and myelocytes are seen in the bone marrow. Otherwise, it resembles the aplastic type, except for the marked hyperplasia of the reticulo-endothelial system. The reticulo-endothelial cells may assume various forms; they may appear as foam cells or clasmatocytes or may assume phagocytic proportions.

5. Infectious mononucleosis:[13] There has been much dispute concerning the sternal marrow findings in this condition. However, it is the opinion of most authors that there is little or no change in the sternal marrow beyond a moderate shift to the left of granulopoiesis. Other workers, such as Freeman (1936), Markoff (1936), and Mallanue (1937), on the other hand, have all found an increase in the cells of the lymphocytic series. Scott (1939), however, seems to think that the findings of the above three workers were due to dilution with the peripheral blood. He says that sternal puncture is of very little value in diagnosing infectious mononucleosis, because even finding a normal marrow, which often occurs, does not exclude lymphoid leukosis.

6. Leukopenic states:[2] Normally neutrophiles are produced in the bone marrow and released in sufficient numbers to maintain the normal number in the peripheral blood. If there be a deficiency of the hypothetical maturation factor or the factor which regulates the release of mature cells into the blood, or if some toxic agent destroys the neutrophiles in the peripheral blood, a leukopenic condition will be present.

Bone marrow studies indicate that in the leukopenic states, the marrow fails to produce a sufficient number of cells rather than their increased destruction in the peripheral blood. If the marrow is subjected to depressing influences, cell development may be altered so that development occurs at a less mature level, or there may be a decrease in the number of mature cells producing the mature types, even though there is no arrest of normal maturation.

Leukopenia may be due to many different conditions, however, it is not the scope of this paper to elaborate on the bone marrow findings in all the conditions producing leukopenia, except to say that in those instances where the primary effect is on the bone marrow, the myelogram reveals a depression of granulopoiesis.

7. Protozoal diseases:[5]

a. Kala-azar: The myelogram in this condition is specific. The marrow is hyperplastic. There is a marked increase in lymphocytes, intense reticulo-endothelial hyperplasia, patchy distribution of platelets, and a marked increase in monocytes in which are found the Leishmania. These consist of a tenuous membrane surrounding a cytoplasm which is faintly basophilic in character, in which minute inclusion bodies are found. These bodies consist of a nucleus which is large and stains deep violet, and centrosomes which are smaller, rod-shaped, and stain moderately basophilic.

Even though the leishmania may be absent in the bone marrow, the myelogram is characteristic. The following table, according to Piney, is a representative myelogram in this condition:

	Per cent
Neutrophiles:	
Myelocytes	18-24
Metamyelocytes	0-1
Polymorphs	8-10
Eosinophiles	0-1
Lymphocytes:	
Small	18-22
Large	18-22
Monocytes	14-18
Plasma cells	1-3

Haemohistioblasts	0-1
Hematocytoblasts	0-1
Normoblasts	3-5
Megaloblasts	1-3

Films show increased cellularity; and platelets are increased.

b. Malaria: Finding the parasites in the marrow cells is not common, but when they are found they are specific. Essentially the marrow findings are monocytic and normoblastic. The monocytic response is more marked in chronic cases than in acute ones. The following table gives a fairly representative picture of the bone marrow in this condition:

	Per cent
Neutrophiles:	
Myelocytes	5-7
Metamyelocytes	0-2
Polymorphs	9-12
Eosinophiles	0-2
Monocytes	10-14
Lymphocytes	2-3
Premyelocytes	0-2
Hematocytoblasts	0-1
Normoblasts	40-50
Macronormoblasts	12-16

8. Tumors:

a. Multiple myeloma:[5] This is the most common type of primary tumor. Although the sternal bone marrow findings are variable, the presence of the so-called "myeloma cells" which make up 3 to 65 per cent of all the cells, is pathognomonic of the condition. The absence of these cells, however, does not rule it out. Even though diagnosis is possible by clinical and radiological methods, changes in the bone marrow take place before peripheral blood changes or clinical and radiological evidence is present.

Most of the cells are of the plasmocytic variety, but myelocytic and erythroblastic responses, though rare, do occur.

There has been much dispute concerning the nature of the "myeloma cell", but whatever the nature of this cell, it appears as a large round or ovoid cell about 15 to 30 microns in diameter. The diameter of the nucleus is from 5 to 7 microns, usually eccentric in position, occasionally nucleolated and leptochromatic, but more often stains densely. Cartwheel arrangements are uncommon. The cytoplasm is basophilic and a pale perinuclear zone may be present; cytoplasmic vacuoles are frequently present. At times 2 or 3 nuclei may be present, but mitotic figures are not seen.

b. Chloroma:[5] The position of this tumor-like condition is far from being clear. Suffice it to say that the bone marrow picture presents the appearance of a leukemia (usually the myeloblastic type) and differs from it through the formation of intra and extra medullary tumor masses. There is a marked tendency for these tumors to invade or erode adjacent structures.

c. Hodgkin's disease:[13] There has been much diversity of opinion among workers in this condition. Most of the authors state that there is no change in the bone marrow beyond a shift to the left of a granulopoiesis with occasional toxic changes. Some workers, Rohr and Hegglin (1936), and Varadi (1938), claim they have found Reed-Sternberg cells in the puncture films, but this has also been disputed by others workers; diagnosis, as before, depends upon the histiological study of an excised lymph node.

d. Storage diseases:[2] The so-called lipoid and non-lipoid histiocytoses (which include Niemann-Pick, Hand-Schüller-Christian, Gaucher) are all really more generalized diseases, but affect the reticulo-endothelial elements of the bone marrow, spleen, as well as other organs, and may produce local areas of myelophthisis and bone rarefaction. The presence of extremely large, mononucleated cells with or without lipoid material in their cytoplasm is diagnostic according to type. Bone marrow biopsies are mainly helpful in confirming rather than making a diagnosis in these conditions.

e. Ewing's tumor:[5] This is a very good example of a tumor that arises from the reticulo-endothelial system of the bone marrow. In the early stages sternal puncture will reveal a normal myelogram, but metastasis from its usual primary focus in the long bones is widespread; and long before there is either clinical or radiological evidence of metastasis, the sternal marrow may contain scattered tumor cells. These cells are large with indented nuclei, irregular hyaline cytoplasm, and resemble histiocytes, possessing all of the characteristics of what are sometimes called dismorphocaryocytes.

Much investigation and study is needed on the reticulo-sarcomas and their allied conditions. With the present knowledge one can make a diagnosis of malignancy or of innocence, but very little can be said about their origin.

f. Secondary tumors:[5] These tumors are much more common than primary growths. It is surprising how frequent tumor cells and signs of hypoplasia can be detected in the absence of clinical and radiological findings. Metastatic tumor cells differ considerably from those found in their primary growth site. In the marrow they are almost always found in groups composed of small and large cells, with a tendency to become much more elongated than epithelial cells elsewhere. Therefore, it is very difficult to determine where the primary site of the tumor is situated.

The myeloid tissue is usually extremely active, thus accounting for the leuko-erthroblastic anemia that may accompany marrow metastasis. But aplasia may occur, and this is often associated with some osteosclerosis.

It is evident that the use of this procedure routinely would indicate that many clinically operable cases have already passed the stage in which cure is possible.

SUMMARY

1. A brief history of bone marrow biopsy has been given.

2. The appearance of the normal myelogram has been described in detail.

3. Three methods of bone marrow biopsy now employed were described: (a) trephine method; (b) aspiration method, and (c) drill method.

By employing the trephine method, one can determine the state of hyperactivity, hypoplasia or defective maturation in one or all of the cell series; whereas in aspirated marrow these matters are questionable, because the cells do not retain their original relationship, probably not their relative proportions, and the material withdrawn is apt not to be representative. Focal lesions are apt to be missed, and aspirated marrow is often unavoidably diluted with blood. Lastly, the cells are more likely to be damaged in preparing blood-marrow mixtures.

However, the aspiration method is simpler, more widely used and repeated biopsies on the same individual may be performed. It is quite satisfactory in certain instances where one does not want to employ the more radical operative procedure of trephining. One should employ both procedures, if necessary, using the trephine method when the more conservative aspiration method fails to give the desired information. The drill method was devised in an attempt to combine the virtues of the trephine and aspiration methods, but as yet is not used very much.

4. Sternal puncture is indicated when: (a) the diagnosis is in doubt and a disease usually showing a typical myelogram cannot be excluded; (b) the etiology of the peripheral blood is obscure; (c) other means of biopsy have failed. It may be also done: (a) to save time if a specific marrow picture is likely to be present; (b) to obtain information concerning prognosis; (c) to determine response to treatment, and (d) determine type of "stem cell" leukemia when this cannot be done from the peripheral blood. It should also be performed whenever splenomegaly is present.

5. The myelogram changes in the more important leukopoietic diseases have been presented with special mention being made of those conditions which produce typical myelograms. Typical myelograms are found in: (a) leukemia, especially acute and aleukemic leukemia; (b) megaloblastic marrow, present typically in pernicious anemia in relapse, achrestic anemia, and sprue; (c) agranulocytosis in the arrested marrow stage; (d) neoplasms: multiple myeloma and metastatic malignancies, especially carcinoma; (e) lipoid histiocytosis; (f) malaria, and (g) leishmaniasis (kala-azar).

CONCLUSIONS

1. Taking all things into consideration, the aspiration method of sternal bone mar-

row biopsy has been found to be the most practical and widely used.

2. Sternal puncture has provided the hematologist with a relatively new and valuable diagnostic procedure which should be employed when it is indicated. It should, however, not be used alone, but in conjunction with other clinical and laboratory data to establish and confirm a diagnosis.

3. One of the greatest handicaps with bone marrow studies is that too few workers are adequately trained to study and recognize intelligently all of the various types of cells found in the bone marrow.

4. Finally, the study and diagnosis of diseases by sternal marrow biopsy is still a relatively new field, and much investigation is needed along these lines before myelogram examination becomes an art.

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THE WAGNER-MURRAY-DINGELL BILLS

These bills, the one introduced in the Senate by Senators Wagner and Murray and a similar one in the House by Representative Dingell, according to newspaper columnists and other press and radio information, seem to have lost considerable amount of support from several of the important groups that were originally very enthusiastic about them. Labor, for example, since Mr. Lewis' demands for a health service to his miners has been granted and since many other unions are making or have been granted similar demands, have not shown the enthusiasm which previously was exhibited. Likewise former military men seem to feel that these bills will subject them to heavy taxation in spite of the fact that the Veterans Administration is apparently prepared to give medical care to all of the soldiers and other members of the armed forces who participated in World War I and in Global War II.

The published reports that are appearing in the Journal of the American Medical Association of the hearings of the Senate Committee on Education and Labor would seem to show that there is considerable amount of sentiment against the bills not only by the medical profession but by other groups. To the medical man the bills seem so ridiculous and the arguments so many against them that it does not seem possible that they could pass an intelligent Congress. However, we never know what may emanate from the Congress and our efforts to defeat the bills should not be relaxed.

One of the interesting features of these bills which rates so high in the thoughts of the medical man is the general lack of interest in them throughout the country. Apparently most people do not even know about the bills as shown by a recent Gallup Poll. These medical insurance bills, according to the poll, were known to only four in every ten persons questioned and the majority of the forty per cent who knew that there were such bills were unfamiliar with their provisions and had given very little thought to how they would affect the general economy of the country. Furthermore of those questioned the majority felt that they would not be willing to pay any more for medical insurance than they are now paying for doctor and hospital bills and about half of those quizzed said they would be unwilling to pay as much.

Lastly, the opinion was expressed by less than half of the people who were questioned, that they would get as good medical service under government supervision as they are now getting. Certainly this poll would indicate that the citizenry of the United States are not particularly interested in the bills nor do the majority of those who know anything about the bills feel that they would get any better medical service than they are now obtaining.

THE ANTI-VIVISECTIONISTS

It is earnestly to be hoped that many doctors may have the opportunity of reading Mr. Bernard DeVoto who writes the section entitled "The Easy Chair" in each month's number of the Harper's Magazine. In the June edition of this long existing and well known publication, he lights into the anti-vivisectionists in a way that the medical profession would hardly have the temerity to do. Mr. DeVoto's expressions and his writings are so superior that we may be pardoned for quoting them rather extensively.

In the first place Mr. DeVoto points out that the so-called anti-vivisection campaigns are carried on by a group of professionals who are in the campaigns not for unselfish purposes, but for what they can get out of them financially. They enlist unpaid volunteers, for the most part "elderly sentimentalists . . . escapees, the perpetually indignant, the perpetually confused . . . They are earnest bewildered souls . . . probably with deflected affections and certainly with all-embracing ignorance and a vigorous resistance to thought." These are the type of people who represent the volunteer workers. What Mr. DeVoto can not understand is why the "front men and women" will sign the literature and manifestos which are prepared by the professionals. "Their zeal is a danger to all of us, but their irresponsibility is a greater danger . . . it is not fair to dismiss them as necessarily either fools or liars—though it is difficult to believe that they are always unaware that the statements they issue and the literature they sign are mendacious, composed almost entirely of misrepresentations, distortions, and misstatements". The essayist goes even further and states that their appearance of benevolence and kindness "covers a pathological misanthropy, a residue of primitive totemism, and a still more primitive destructiveness". He contends that the pathologic champion of animals is a pathologic hater of men.

Mr. DeVoto states that to answer these people is both foolish and impossible. Probably every honorary officer of an anti-vivisection society owes his life or that of a member of his family to medical and surgical experiments on dogs. He points out that what we know of physiology has been learned almost entirely from animal experimentation; almost all advances in surgical technic have come from experiments on animals; what we know about immunity and about the use of vaccines and antitoxins and serums has been learned from the same source. When a sulpha drug is given, or penicillin, or even a barbiturate, when a woman and her infant survive a difficult delivery the sedative and anesthesia and the surgical technic are the result of animal investigation. He writes that "one of the few undebatable triumphants of civilization, progress in medical science, rests on animal experimentation."

In his concluding paragraph he notes that at times there appear small religious sects in the desperately uneducated which in their superstitious ignorance often show a compulsive frenzy which seems diseased. "The charity that excuses ignorance can not be extended to them (the anti-vivisectionists), for mostly they are well educated and economically fortunate above the average. They must be thought of as at best neurotic and at worst corrupt."

DIAGNOSIS OF CANCER

One of the most fascinating chapters in the book of diagnosis has to do with the recognition of cancer as developed by the technic of Dr. George N. Papanicolaou.* To recall his original observations which appeared some years ago, he showed that diagnosis of cancer of the female genitalia might be made early in the course of the disease through the study of vaginal smears. Very little interest was aroused in his observation until within the last few years. Now, however, it has become part of the routine of the periodic health examination to have vaginal smears made, knowing that malignancy of the genital tract may be recognized through the appearance of pathologic cells in the smear more readily, more promptly and earlier than by any other method. Furthermore any woman who has symptoms suggestive of carcinoma of the uterus should have a vaginal smear made by her physician and studied by a competent pathologist.

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*Papanicolaou, George N.: Diagnostic value of exfoliated cells from cancerous tissue, J. A. M. A., 131:372, 1946.

The fact that the malignant cells exfoliate from the uterus is now well recognized but as a recent paper of this well known investigator shows, it is not generally accepted that malignancy may be recognized elsewhere in the body early in the course of the disease through the findings of pathologic cells in the excretions or secretions. A careful study of the sputum may show abnormal cells which may lead to the diagnosis of bronchiogenic carcinoma before the cancer can be visualized by the endoscope. The gastric contents may show cancer cells very early in the course of the development of the malignancy. The urinary sedimentation will do the same. In fact the diagnosis of early carcinoma may be made by the study of the smears from the secretions and excretions with proper technic, in a large number of cases. It is to be hoped that the studies of Dr. Papanicolaou will be carried out extensively in laboratories throughout the country. Certainly the results that the author has had are remarkable but as he says they are still far from being complete and it will be many years before the potentialities of this new diagnostic approach have been thoroughly explored by many investigators.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

EMIC PROGRAM

With further reference to termination of the EMIC Program by the State Society the Executive Committee at a meeting on June 8 ruled that ANY CASE PREGNANT BEFORE THE TERMINATION OF THE EMIC PLAN (MAY 8, 1946) MAY BE ACCEPTED BY MEMBERS OF THE SOCIETY.

SENATE BILL 2143

Special attention is called to Senate Bill 2143, recently introduced by Senator Taft for himself, Senator Ball and Senator Smith, known as the "National Health Bill". This bill developed as a result of differences of opinion on the Wagner-Murray Bill S.1606, and might be offered as a substitute to the Committee on Education and Labor to which has been assigned S.1606. The secretary has had occasion to review this bill and finds the basic principles to be very sound. There are a few necessary amendments which should be made to meet the full requirements of the medical profession in their relation to the public on any prepayment plan. A very fine review of this bill recently occurred in the Journal of the American Medical Association (May 25, 1946, Page 338).

COUNCIL ON MEDICAL SERVICE AND PUBLIC RELATIONS

At the meeting of the Executive Committee on June 8 plans for a Council on Medical Service and Public Relations in the State Society which had been approved by the House of Delegates were discussed. It was decided that the president should appoint a committee to work with him in planning details for such a council; these to be submitted to the Executive Committee for final action. It was also agreed that Dr. Peacock, as delegate to the A.M.A., should be requested to represent our Society at a meeting of state chairmen of councils on medical service and public relations to be held in San Francisco during the time of the A.M.A. meeting and that he be instructed to submit a report to the special committee on his return.

PREPAYMENT MEDICAL SERVICE

Dr. O. B. Owens, Chairman of the Committee on Prepayment Medical Service presented to the Executive Committee a schematic diagram of the proposed set-up of the Louisiana Physicians Service, Inc., and asked for approval of the following, which was granted: 1. Material as presented in rough draft as a guiding principle for further work by the committee and the attorney. 2. Authorization for the Board of Directors to employ Mr. Barret (of Shreveport) or some other well-qualified insurance attorney, to draw the necessary legal documents and serve as attorney for the Board in the organization and early period of operation of the plan; the fee for which would be paid as an organization expense by the Corporation. 3. Authorization for the proper officers of the Louisiana State Medical Society to make available for deposit with the State of Louisiana Insurance Department the necessary initial deposit, together with the sum of $10,000 for implementation of the organization and operation of the plan; this amount to be made available upon submission by the attorney of the necessary loan agreement, as indicated in the documentary outline. 4. Authorization for the Board of Directors to enter into contract negotiations with local hospitalization units throughout the state or other organizations or individuals for administration of the plan in specified areas of the state. 5. Authorization of the use of press, radio, pamphlet or other forms of advertising by the Board and its members, including a statement that the plan is sponsored and approved by the Louisiana State Medical Society and its constituent bodies and members. 6. Authorization for the Board to prepare copy of printed matter and the taking of bids and awarding of contracts for printing of the necessary policies, contract forms, stationery and advertising material. 7 Authorization for the Board to employ either on a temporary or permanent basis, as later need may indicate, a trained surgical plan executive. 8. Authorization for the Board, through its contractive representatives, at the proper time as defined by the Insurance Department, to begin soliciting and enrolling subscriber groups.

It has been further decided that the headquarters for the Louisiana Physicians Service, Inc. will be in the domicile of the State Medical Society in New Orleans.

Dr. Owens and his committee are to be congratulated on the excellent service they have rendered to our organization in consideration of plans for voluntary prepayment medical service in the state.

LOUISIANA LEGISLATION

The Committee on Public Policy and Legislation has been extremely active during recent weeks in connection with proposed legislation which will affect the medical profession of this state. Outstanding in their accomplishments is success in having the Senate chiropractic bill defeated and a similar bill in the House withdrawn. Also House Bill 38, in re temporary license to foreigners to practice medicine in Louisiana, which the House of Delegates at the annual meeting went on record as opposing, was after much effort on the part of our legislative committee, defeated.

The members of this important committee are still giving much time and thought to other legislative matters to be considered in the future and much gratitude should be expressed to them for their willingness to assist organized medicine in this manner.

RESOLUTION IN RE CHIROPRACTORS

The following resolution has been sent to each component society in the state and is published here so that members may report to their local organizations such illegal practice of which they have knowledge in their parish: Be it resolved by the Executive Committee of the Louisiana State Medical Society in session in New Orleans on June 8, 1946, that the parish and district medical societies of the State of Louisiana be authorized to request of the Louisiana State Board of Medical Examiners that they start legal proceedings against any and all chiropractors now applying their supposed profession in opposition to our present medical laws. Senate Bill No. 66 of the 1936 Legislature which would legalize chiropractors in this state was voted down by the Committee on Education and Quarantine on June 5. There was only one dissenting vote. Most of the co-authors of the bill when they found out that the medical profession was against this bill were in favor of withdrawing the bill. We are now assured that the state legislature and representatives of the people of the state do not want chiropractors attempting to practice medicine here. Be it further resolved that a copy of this resolution be sent to every parish and district medical society in the state advising them to request the State Board of Medical Examiners to start legal proceedings against all illegal practitioners as soon as possible. Be it further resolved that the State Medical Society also request the State Board of Medical Examiners to assist in prosecuting these men wherever necessary.

1947 ANNUAL MEETING

The dates of May 12-14, 1947 have been selected for the meeting of the State Society to be held in New Orleans next year and the president, Dr. Rhett McMahon, has appointed the following members to serve as chairmen of scientific sections for the meeting: Dr. John L. Beven, Baton Rouge, Bacteriology and Pathology; Dr. R. Phillips, Baton Rouge, Ear, Nose and Throat; Dr. Lionel F. Lorio, Baton Rouge, Eye; Dr. James E. Toups, Baton Rouge, Gastro-enterology; Dr. Henry C. Hatcher, Baton Rouge, General Surgery; Dr. W. D. Beacham, New Orleans, Gynecology; Dr. Phillip H. Jones, New Orleans, Medicine and Therapeutics; Dr. Arthur D. Long, Jr., Baton Rouge, Nervous Diseases; Dr. Paul W. Winder, Shreveport, Obstetrics; Dr. T. Jeff McHugh, Baton Rouge, Orthopedic Surgery; Dr. David S. Miller Baton Rouge, Pediatrics; Dr. Bernard E. Nelken, Baton Rouge, Public Health and Sanitation; Dr. Lester J. Williams, Baton Rouge, Radiology; Dr. John G. Menville, New Orleans, Urology.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

July 8. Scientific Meeting, 8 p. m.

July 17. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

July 18. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

July 24. Catholic Physicians' Guild, 8 p. m.

July 25. De Paul Sanitarium Staff, 8 p. m.

July 26. L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Ochsner Clinic Staff, 8:30 p. m.

July 31. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Aug. 1. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Executive Committee, Baptist Hospital, 8 p. m.

Aug. 5. Board of Directors, Orleans Parish Medical Society, 8 p. m.

Every Tuesday, Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

Every Wednesday, Medical Discussions, Radio Station WDSU, 9:15 p. m.

Dr. Samuel Barkoff gave a paper on "The Psychoanalytic Treatment of Bronchial Asthma" at the Washington-Baltimore Psychoanalytic Society meeting in Baltimore on May 12.

Dr. Ernest C. Faust addressed the 27th meeting of the American College of Physicians in Philadelphia, May 17.

Dr. Hiram W. Kostmayer was recently elected to honorary membership in the Tulane History of Medicine Society.

Dr. Lucien A. LeDoux recently attended the forty-seventh annual meeting of the American

Therapeutic Society in Atlantic City, May 11-12. Dr. LeDoux is second vice-president of the society.

Dr. Alton Ochsner recently addressed Loyola university students on the topic "Effects of Tobacco on the Human Body." The lecture was sponsored by Beta Epsilon Upsilon, national honorary medical technology society at Loyola.

Dr. Thomas Benton Sellers attended the mid-year clinics of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons held in Chicago recently.

Dr. H. Ashton Thomas was appointed a member of the Board of the City Health Department; Drs. Boni J. DeLaureal and John M. Whitney were re-appointed.

Dr. Julius Lane Wilson was re-elected president of the Louisiana Tuberculosis Association at the annual meeting of that organization in Alexandria, May 5. Other members of this Society elected to office were: Drs. Sydney Jacobs and Maurice Campagna, vice-presidents; Drs. J. A. Danna, Edgar Hull, John H. Musser, I. L. Robbins, Suzanne Schaefer and John M. Whitney, members of the executive committee.

Congratulations to Dr. and Mrs. Arthur Calix on the arrival of Kathleen Elizabeth May 6.

MEDICAL TOASTMASTER

Dr. Edwin L. Zander was recently elected president of the Medical Toastmasters. Other officers elected were: Dr. Philip H. Jones, vice-president; Dr. Lawrence J. O'Neil, secretary-treasurer; Dr. J. A. Colclough, sergeant-at-arms; and Dr. Charles A. Bahn, deputy.

WELCOME HOME

Dr. Norton M. Voorhies was recently separated from military service. He has resumed practice at 1712 Pere Marquette Bldg.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

NEW DEAN OF L. S. U.

The board of supervisors of Louisiana State University announce the appointment of Dr. Vernon William Lippard of Columbia University as dean of the university medical school. Dr. Lippard is a graduate of Yale Medical School and for several years was a member of the Cornell University faculty. He was in the army with the rank of lieutenant colonel and since his release from military service has been associate dean of the medical school at Columbia University. He is one of the well known younger pediatricians in this country being certified by the American Board of Pediatrics and a member of the Society for Pediatric Research as well as county, state and national medical organizations. He is a member of Alpha Omega and Sigma Xi honorary societies.

Dr. Lippard will succeed Dr. George W. McCoy who has been acting dean since the resignation of Dr. Wilbur C. Smith.

LAKESHORE HOSPITAL

The new Lakeshore Hospital was opened for patients on Saturday, June 1. On Sunday, June 2, a reception was given to the medical profession and others at which time the hospital could be inspected and the arrangements that had been made to take care of the patients were shown to the visitors. The hospital is conducted by the New Orleans Medical Foundation, most of whom are physicians who have served in the armed forces. Dr. Charles B. Odom is president of the Foundation and to him as well as to others of the officers a great deal of credit should be given for obtaining these additional hospital beds so badly needed in New Orleans. The Lakeshore Hospital is open to all members of the medical profession belonging to the Orleans Parish Medical Society and promises to help relieve the exigency for beds in the city. Mr. Evert E. Moody is director of the new institution.

NEW CHARITY HOSPITAL APPOINTMENTS

Dr. Roscoe L. Pullen, who for the past several years has been assistant director of the New Orleans Charity Hospital, resigned effective July 1. To replace Dr. Pullen, Dr. O. P. Daly, director of the institution, has appointed Dr. Jacques Magne, who will be at the head of the medical division of the hospital and Dr. Joseph Dills, who will have charge of the surgical division.

Dr. Magne served with distinction throughout the war, obtaining the rank of lieutenant colonel. He is a New Orleanian who graduated from Tulane in 1938 and became a resident on the medical service until called to active duty in 1941.

Dr. Dills is a graduate of the Washington University in St. Louis and since his graduation in 1941 he has been connected with the Charity Hospital, first as an intern then as admitting physician and finally as surgical resident. He was appointed on the L. S. U. surgical staff on July 1, 1943.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly has elected the following members for office for the year 1946-1947: Dr. Joseph S. D'Antoni, President-elect; Dr. Randolph Lyons, First Vice-president; Dr. H. Ashton Thomas, Second Vice-president; Dr. C. S. Holbrook, Third Vice-president; Dr. Max M. Green, Secretary; Dr. Edwin H. Lawson, Treasurer; Dr. Barrett Kennedy, Director of Program; Dr. J. O. Weilbaecher, Jr., Asst. Director of Program; Dr. Ambrose H. Storck, Asst. Director of Program. Executive Committee: Dr. Howard R. Mahorner (two year term), Dr. Lucien A. Fortier (two year term).

The following officers elected in 1944 who will also serve for the coming year are as follows: Dr. William H. Gillentine, President. Executive Committee: Dr. L. C. Chamberlain, Retiring President; Dr. Frederick F. Boyce, Dr. Charles B. Odom.

DE PAUL SANITARIUM

The regular monthly meeting of the Medical Staff of De Paul Sanitarium was called to order at 8:00 p.m. on May 23, 1946, with Drs. Friedrichs, May, Otis, Beach, Barkoff, Dupuy, Hunter Harris, Holbrook, Anderson, H. Colomb, Hite, Connely, Monroe and Dubos in attendance.

The scientific program began with a discussion by Dr. Holbrook of Case No. 4646, that of a white female approximately five feet two inches in height, weighing 287 pounds, first seen in 1923 and again in 1930. At that time she was suffering from depression and melancholia. One month ago she was again seen for the same symptoms and admitted to De Paul, but because of her extreme obesity and hypertension she was considered a poor risk for electroshock and therapy was limited to simple palliative measures. She was placed on a low caloric diet and given thyroid medication. With good nursing care she showed sufficient improvement to leave the institution and return home within one month's time. The case was further discussed by Drs. Barkoff, Otis and Dubos.

Dr. Beach next summarized briefly the properties, dangers and uses of curare in relationship to shock therapy. This topic was further discussed by Drs. Holbrook and Anderson.

At the request of the president of the staff the minutes of the previous meeting were read and after due motioning and seconding were adopted as read.

Dr. Otis as Chairman of the Records Committee read the reports of the various departments following which Dr. Friedrichs, Chairman of the Library Committee reported additional donations to the Lewis A. Golden Collection Fund.

There being no further business the meeting adjourned at 9:10 p.m., followed by the serving of refreshments.

Louis J. Dubos, M. D., Secreary.

LOUISIANA PHYSICIANS PARTICIPATING IN THE SCIENTIFIC PROGRAM OF THE A. M. A. MEETING

The following doctors from Louisiana have presented papers or exhibits at the annual meeting of the A. M. A. which started Monday, July 1, in San Francisco.

In the general scientific meeting Dr. Alton Ochsner, professor of Surgery at Tulane Medical School, gave a lecture on thrombosis and embolism and participated in the discussion on the problem of phlebothrombosis and thrombophlebitis. Colonel M. E. DeBakey of New Orleans, was one of the joint authors on the use of streptomycin in clinical practice. Dr. Champ Lyons, Professor of Clinical Surgery at Tulane Medical School, read a paper on chemotherapy in wound management. Dr. Ralph V. Platou, Professor of Pediatrics at Tulane Medical School, addressed the section on dermatology and syphilology on penicillin therapy in early congenital syphilis. Two New Orleans doctors appeared before the section on gastroenterology and proctology, Dr. Donovan C. Browne, Assistant Professor of Medicine at Tulane, discussed the treatment of intestinal conditions as based on disordered functions, and Dr. D. N. Silverman, Professor of Clinical Medicine at L. S. U. Medical School and Dr. Alan N. Leslie had a paper on intestinal tumors of dysentery origin.

The Chairman's address before the section on anesthesiology was given by Dr. Ansel M. Caine, Assistant Professor of Surgery at Tulane Medical School. In this same section Dr. John Adriani, Clinical Assistant Professor in anesthesia at L. S. U. Medical School, was one of a group of authors who spoke on subarachnoid use of vasoconstrictors in spinal anesthesia.

At the section exhibits, Dr. George E. Burch, Associate Professor of Medicine at Tulane Medical School and Dr. Travis Winsor, formerly on the staff of Tulane Medical School, and Dr. James L. Kimball had an exhibit on the clinical applications of venous pressure measurements. Dr. John Adriani and Dr. Ray T. Parmley had an exhibit from Charity Hospital and Louisiana State University School of Medicine on saddle block anesthesia. Dr. Guy A. Caldwell, Professor of Orthopedic Surgery at Tulane Medical School, and Dr. Donald Imrie of the Ochsner Clinic, had a motion picture on the analysis of muscle function in poliomyelitis. Colonel Roy H. Turner, Associate Professor of Medicine at Tulane Medical School, participated in the Medical Consultants Division of the U. S. Army, his exhibit showing diagnosis, course and treatment of infectious hepatitis.

NEWS ITEMS

Dr. Ernest Carroll Faust has received a Medal of Freedom for the work which he has done on schistosomiasis. It may be recalled that Dr. Faust was invited by the government to make a study of the problem of schistosomiasis in the Phillipines and western theatre and spent some months in this area.

Dr. Alton Ochsner has been selected as the president-elect of the American Association for Thoracic Surgery.

Dr. Wilbur C. Smith has been appointed director of student health and physical activities at the University of Wyoming.

PHYSICIAN WANTED

A physician is needed in Franklin, Louisiana to take over practice of a physician recently deceased. Office space and equipment and also records of the former doctor are available. For further information contact the office of the State Medical Society.

MISSISSIPPI VALLEY MEDICAL SOCIETY

The eleventh annual meeting of the Mississippi Valley Medical Society will be held at the Hotel Jefferson, St. Louis, September 25-27.

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE

Will hold its twenty-fourth annual scientific and clinical session September 4, 5, 6 and 7, inclusive, at the Hotel Pennsylvania in New York. Scientific and clinical sessions will be given each day. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction courses will be held September 4, 5, and 6. These courses will be open to physicians and to therapists registered with the American Registry of Physical Therapy Technicians. For information concerning the convention and the instruction course, address the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

INTERNATIONALLY KNOWN SPEAKERS AT COLLEGE OF SURGEONS ASSEMBLY IN DETROIT

Mr. Hamilton Bailey of London, England, will visit Detroit in October to deliver an address at the Eleventh Assembly of the United States Chapter, International College of Surgeons. His subject will be "Impending Death under Anesthesia".

Other eminent speakers and clinicians who will appear at the I. C. of S. clinics, and the Assembly to be held in Detroit's Masonic Temple, October 21, 22 and 23, include: Dr. Albert Jirasek of Prague, Dr. Francisco Grana of Lima, Peru, Dr. Lyon Appleby, Vancouver, B. C., Dr. Felipe Carranza of Buenos Aires, Dr. W. Wayne Babcock of Philadelphia, Dr. R. W. McNealy, Chicago, Dr. Wm. C. MacCarty, Rochester, Minn., Dr. Edwin L. Zander, New Orleans, Dr. Rudolph Nisson, New York, Dr. Max Thorek, Chicago, Dr. H. E. Billig, Los Angeles, Dr. S. W. Harrington, Rochester, Minn., and others.

Copy of program and detailed information, including housing arrangements, may be obtained by writing Dr. L. J. Gariepy, Secretary, 16401 Grand River Avenue, Detroit 27, Michigan.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The annual meeting of the Board was held in Chicago, Illinois, from May 5 to May 11, 1946, at which time one hundred forty-one candidates were certified.

A number of changes in Board regulations and requirements were put into effect. Among these is the requirement that case records must now be forwarded to the Secretary's Office from thirty to sixty days after the candidate has received notice of his eligibility for admission to the examinations for certification. At this meeting the Board also ruled that it will not accept the nine months' residency as an academic year toward years of training requirements following the termination of the official period of intern and residency acceleration, April 1, 1946.

The next written examination (Part I) for all candidates wil be held in various cities of the United States and Canada on Friday, February 7, 1947 at 2:00 p. m. Candidates in military service are requested to keep the Secretary's Office closely informed of changes in address.

Applications are now being received for the 1947 examinations. Closing dates for these applications will be November 1, 1946.

For further information and application blanks address Paul Titus, M. D., Secretary, 1015 Highland Building, Pittsburgh, 6, Pennsylvania.

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ARMY LOWERS DISCHARGE REQUIREMENTS FOR MEDICAL CORPS OFFICERS

Discharge requirements for Medical Corps general duty officers were reduced on May 1 from 39 to 30 months service and from 45 to 39 months for certain specialist officers.

Requirement on points remains at 60 and the age limit at 45 years.

This move will result in the discharge of approximately 2,000 doctors and will leave about 3,000 Medical Corps officers not included in the ranks of the Regular Army, volunteers and Army Specialized Training Program graduates.

Officers returning from overseas will be discharged automatically if they would become eligible for discharge within the following six months instead of four months as previously stipulated. Also, Army doctors overseas regardless of their military occupational specialty classification, will be separated or enroute to the United States within 60 days of date of eligibility.

That the Army is demobilizing its Medical Corps officers as quickly as possible without jeopardizing the treatment of patients is shown in the announcement that since VE Day about 32,000 have been discharged.

The specialists who require the longer term of service for discharge are gastroenterologists, cardiologists, urologists, dermatologists, anaesthetists, general surgeons, physical therapists, radiologists, pathologists, orthopedic surgeons, internal medicine specialists and eye, ear and nose specialists.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending April 27 showed measles leading all other reportable diseases with 285 cases. Following in numbers greater than ten were hookworm infestation 57, pulmonary tuberculosis 50, influenza 47, malaria 35, mumps 33 and septic sore throat 26. There was one case of poliomyelitis reported this week in Webster Parish. There is something peculiar about the reporting of some of these diseases. In the previous week there were only two cases of cancer listed and six of unclassified pneumonia whereas for the week ending May 4 there are 193 cases of unclassified pneumonia and 166 incidences of cancer. Other prevalent diseases include 119 cases of measles, 60 of pneumococcic pneumonia, 40 of whooping cough, 38 of mumps, 28 of pulmonary tuberculosis, 21 of hookworm infestation, 15 of scarlet fever, 13 of amebiasis and 12 of malaria. Of the rarer diseases, poliomyelitis was reported in one instance in Orleans Parish. For the week which came to a close on May 11 the six diseases which occurred in numbers greater than ten were measles 190 cases, pulmonary tuberculosis 48, whooping cough 25, mumps 23, unclassified pneumonia 19 and German measles 17. This week there was another case of poliomyelitis, this time listed from Lafayette Parish. For the week which ended May 18 the report shows obviously that measles was still quite prevalent, there being 165 cases listed, followed by 40 of pulmonary tuberculosis, 36 of mumps, 29 of malaria, 20 of unclassified pneumonia and 12 each of whooping cough and septic sore throat. There were five cases of poliomyelitis listed this week, no two of which originated in any one parish. For the week which closed May 25 there were only 64 cases of measles listed, 27 of pulmonary tuberculosis and 18 of mumps. No other disease occurred in number greater than ten. Again, there were five cases of poliomyelitis reported in the state.

The Bureau of Epidemiology of the Louisiana State Board of Health now reports venereal disease incidence as a separate report. For the month of March there were listed 2,091 cases of gonorrhea, 1,793 of syphilis, 64 of chancroid, 32 of granuloma inguinale and eight of lympopathia venereum. For the month of April there were listed 1,795 cases of syphilis, 1,656 of gonorrhea, 48 of chancroid, 30 of granuloma inguinale and eight of lymphopathia venereum.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that for the week ending May 11 there occurred in the City of New Orleans 133 deaths divided 85 white and 48 colored. Of this 133, 11 were children under one year of age. The following week showed a very marked decrease; the total deaths being only 102 as contrasted with the previous three year average of 133. Of these deaths 67 were in the white population, 35 in the colored and seven were in infants. There was a slight increase in the number of deaths for the week ending May 25 but this was still below the three year average. The deaths were apportioned 67 white, 52 colored and the total number of deaths in children was only seven. For the week which closed June 1 the remarkably low death rate was still maintained. There were only 117 deaths in the city, 69 of which were in the white population and 48 in the colored but the infant deaths had risen to 18. The death rate for the week ending June 18 was higher than for the previous week but still below the three year average. In this particular week the 131 deaths in the city were divided 74 white, 57 colored and 11 infants.

CORRECTION

In the article by Robert A. Katz, M. D., "Impending Ischemic Gangrene" in the June issue, page 551, paragraph one, beginning with line 13, reads as follows:

Within three days, despite the fact that the lower extremity is kept in a dependent position for a period up to three hours, the ulcer on the lower half of the leg has decreased about one-half in size and the portion which has not been covered by epithelium is covered with healthy granulation tissue.

This should read: Within three days the pain was entirely relieved and has not returned despite the fact that the lower extremity is kept in a dependent position for periods up to three hours. The ulcer on the lower half of the leg has decreased about one-half in size and the portion which has not been covered by epithelium is covered with healthy granulation tissue.

WOMAN'S AUXILIARY

LUNCHEON FOR WIVES OF RETURNING DOCTORS

In honoring the doctors who have recently returned from service in the armed forces, Mrs. M. C. Wiginton, press and publicity chairman of the Woman's Auxiliary, State Medical Society, gave a delightful luncheon at her home. Wives of the doctors were special guests and a number of out-of-town ladies were in attendance, including Mrs. Arthur Long of Baton Rouge, state president of the auxiliary, and Mrs. F. U. Darby, also of Baton Rouge, president-elect of the East Baton Rouge Auxiliary. Mrs. Roy Carl Young of Covington, who is a past president of the Woman's Auxiliary of the State Medical Society, was also present. She is now serving as councilor for the sixth district. Mrs. Young was accompanied by Mrs. H. E. Cannon of Covington.

Those attending from Tangipahoa Parish were as follows: Mrs. Glenn T. Scott and Mrs. Collins P. Lipscomb of Ponchatoula; Mrs. DeLoach Thames, Mrs. C. D. Alessi, Mrs. S. J. Cali, Mrs. A. J. Feder, of Hammond; Mrs. Jesse H. McClendon of Amite.

Following the luncheon Mrs. Long and Mrs. Young gave interesting talks on the work being done by the auxiliaries and the importance of organizing, especially now when everyone is vitally interested in the proposed Murray-Wagner-Dingell bills and others of similar note, which means enforced socialized medicine on the general public. It was pointed out that through close contact with the medical profession the auxiliaries realize the danger of such bills being imposed upon the people, hence feel willing to do everything possible to assist in securing the proper information regarding the danger of such legislation.

Wives of doctors of the parish agreed to reorganize and the following officers were elected: President, Mrs. Glenn T. Scott of Ponchatoula; President-elect, Mrs. DeLoach Thames of Hammond; Vice-president and Program Chairman, Mrs. S. J. Cali of Hammond; Secretary, Mrs. Collins Lipcomb of Ponchatoula; Treasurer, Mrs. C. D. Alessi of Hammond.

Various chairmen are to be appointed by the president later, it was explained. It was stressed that the objects of the auxiliary are to interpret the aims of the medical profession to other organizations interested in the promotion of health education, to assist in the entertainment of the medical society, to encourage friendliness among families of the medical profession, and to do work approved by the advisory council of the American Medical Association. It was pointed out that the wives of doctors working together as an auxiliary could serve their respective communities efficiently and make community life much better. The ladies present all expressed a desire that they be called upon whenever help is needed in any of the communities of Tangipahoa Parish.

Reports from over the state concerning the activities of the auxiliaries should be made to Mrs. Wiginton at Hammond, in her capacity as press and publicity chairman of the Woman's Auxiliary to the Louisiana State Medical Society.

TEA FOR MRS. ARTHUR LONG

Honoring Mrs. Arthur Long recently at the Woman's Clubhouse in Baton Rouge were members of her own auxiliary and guests from out of town including groups from New Orleans and surrounding communities. Guests arrived at the clubhouse between the hours of three and five o'clock, being greeted at the doorway by Mrs. Rhett McMahon, whose husband is the newly-installed president of the State Medical Society and Mrs. B. E. Nelken. Guests were registered by Mrs. Eugene Toups. The tea tables were presided over by Mrs. Lionel Lorio, Mrs. J. Webb McGehee, Mrs. Lester J. Williams and Mrs. Henry Voss.

Heading the receiving line was Mrs. Charles E. Allen, Jr., the new president of the Baton Rouge Auxiliary, followed by Mrs. Long, Mrs. Frank Jones, state corresponding secretary, Mrs. Russell Kuehnle, state treasurer, Mrs. John L. Beven and Mrs. Felix Boizelle, general chairmen of the tea, Mrs. F. U. Darby, president-elect of the Baton Rouge Auxiliary, and Mrs. Cecil Lorio. Mrs. Wiley A. Dial was in charge of the tea tables. Miss Celeste Jamerson played a soft accompaniment at the piano throughout the afternoon.

The group of tea girls who served during the afternoon included Mrs. Guy Riche, Jr., Mrs. George Thomas, Mrs. Sterling Albritton, Mrs. McHugh Simmons, Mrs. William Cook, Mrs. Courtland P. Smith and Mrs. Claude McConnell.

Out of town guests at the tea included Mrs. Carroll F. Gelbke, president of the New Orleans Auxiliary, Mrs. James W. Warren, president-elect of the State Auxiliary, Mrs. Paul G. Lacroix, past president of the State Auxiliary and Mrs. J. S. Dunn, Mrs. C. Grenes Cole, Mrs. J. Kelly Stone, Mrs. C. L. Peacock and Mrs. Roy B. Harrison, all of New Orleans; Mrs. Rhodes Spedale, Plaquemine, past state president; Mrs. Charles J. Wise, Angola; Mrs. William Roberts, Clinton; Mrs. Emerson W. Singletary, Baker; Mrs. D. B. Barber and Miss Jane Hall, of Alexandria.

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BOOK REVIEWS

Principles of Dynamic Psychiatry, Including an Integrative Approach to Abnormal and Clinical Psychology: By Jules H. Masserman, M. D. Philadelphia, W. B. Saunders Co. 1946. Pp. 322. Price $4.00.

Just a glance through this volume discloses some of its brilliance, and deeper study of it reveals its profound knowledge. The author wrote it to get into print certain fundamentals of modern dynamic psychiatry, heretofore being taught only through lectures and percepts, so he could divert his efforts to clinical teaching and guidance to which reading alone must forever remain supplementary.

In Part I, Chapter 6, it is related that while affective, regressive and schizophrenic defense mechanisms are therein grouped together not because of their close relationships or similarity, but for the reason that when pervasive and excessive they give rise to definite types of behavioral aberrations and such reactions also have their roots in various "normal" modes of adaptive behavior.

On page 66—Cases: Acute regression, combat neurosis—appears an excellent example of an illustrative material note: "A soldier, however well-disciplined and self-reliant, if subjected overlong to physical exhaustion and the intolerable hell of combat and carnage in the ever-present shadow of death, will eventually reach his limit of tolerance and will thereafter show a gradual dissolution of adult habit-patterns and an accelerating return to childlike and then infantile behavior. First his intellectual interests and activities will become dulled and his affective reactions progressively more irritable and primitive; concurrently, he will begin to neglect habits of personal hygiene, even to the point of careless evacuation and soiling. Next, his fantasies may become preoccupied with hazy dreaming of the comforts and securities of his own home; finally, in sudden desperate denial of all mature considerations of prestige, duty, discipline or even physical danger, he may abandon his weapon, cry out against all about him and fall to the earth sobbing piteously and inconsolably for his mother. Obviously, during this extreme reaction, threats of further discipline or even death are useless; the soldier must be evacuated as a psychiatric casualty, permitted for the time being to cling emotionally to some kindly therapist as a parent surrogate, and thereafter treated by every means at hand to restore his self-confidence, his group loyalties and his normal defenses against anxiety, before his regression becomes fixed at the level of phobia-ridden, helpless passivity."

The purposes of this work are, then, to provide an orientative introduction to the principles of modern dynamic psychiatry, to outline their application to the technics of clinical diagnosis, and to demonstrate the rationale and methods of effective therapy. This book is a most notable, important contribution to medical science and, if there is a better one, the reviewer has not seen it.

C. P. May, M. D.

Voies et Centres Nerveux: By J. Delmas and A. Delmas (both of the Faculty of Medicine at Montpellier). Paris, Masson et Cie., 1946. Pp. 164, figs. 92. 270 fr.

This little paper-bound volume, published in the series "Libraires de L'Académie de Médecine," is offered as an anatomic-physiologic introduction to neurology. In keeping with the objectives of such an introduction, the subject matter is condensed and simplified. The organization is well planned for the purpose of correlating anatomy and functional relationships. The illustrations, all in the form of simple line drawings, are judiciously selected and instructive. The book contains a few footnote references to original literature. They represent contributions of French authors only, even for the period before the war.

Harold Cummins, Ph.D.

Physical Diagnosis: By Ralph H. Major, M. D. Philadelphia, W. B. Saunders Co., 1945. 3rd ed. rev. Pp. 444. Price $5.00.

By now Major's Physical Diagnosis has established itself solidly as a worthwhile text book in teaching this important subject. A third edition seems to bear out this opinion and a perusal of the book is quick to give the reason for its success. This third edition presents no tremendous changes in the contents of the former two. Additions, alterations, and changes in former points of view, of a minor but nevertheless important nature, characterize this latest book. When a publication has been before the profession for nine years, it is no longer necessary to detail its contents. It is still with all its defects the best modest sized volume that we have at this time.

I. L. Robbins, M. D.

The Extremities: By Daniel P. Quiring, Ph.D. Lea and Febiger, Philadelphia, 1945. Pp. 117, 106 engravings. Price, $2.75.

This small book consists of diagrams of the muscles which act on the extremities. No attempt is made to show the relationship of the muscles to each other. However, the nerve supply, blood supply and primary function are given in addition to the principal origin and insertion.

Although this book will serve those who wish to review anatomy or those who wish to refresh their memories concerning a particular muscle, it does not contain enough about each muscle to make the review comprehensive. The fact that the motor point of each muscle is given should make it useful to those who are learning muscle stimulation tests and those who do them infrequently.

The references to the standard texts, Gray and Cunningham, have been carefully kept and should assist the reader to find the details which have been missed in this series of drawings. This book can be recommended to those who wish a hasty review and to those who have need to learn the motor points of muscles.

Fred S. Marshall, M. D.

Clinical Parasitology: By C. F. Craig and E. C. Faust. Lea and Febiger, Philadelphia, Pa. 4th ed. 1945. Pp. 871, 305 engravings and 4 colored plates. Price $10.00.

The fourth edition of this well established book has been enlarged somewhat over the previous edition. The section on arthropods and human disease now includes a more comprehensive consideration of the arthropod-borne diseases as well as of their vectors. In most instances the authors have, with characteristic thoroughness, incorporated the important recent contributions and advances in the field of medical parasitology during the war years up to the time of publication. To mention a few examples, it includes information derived from the experience of the armed forces with filariasis, recent data on dengue and sandfly fever, and mentions the uses of the insecticide DDT in insect control and the improved insect repellents. This edition unfortunately does not note the change in dosage of atabrine for the treatment of malaria which resulted from studies of the pharmacology of the drug and recognition of the relation of the plasma level of atabrine to its therapeutic activity. However, it is pointed out in the text that suppressive atabrine therapy with proper dosage has been found adequate to cure practically all *P. falciparum* infections but is not similarly effective for *P. vivax*. The final section which contains important references to the literature in clinical parasitology is exceedingly helpful for the student or individual who desires to seek more information on any particular subject. It is an excellent and authoritative coverage of the subject of clinical parasitology.

J. C. Swartzwelder, M. D.

Common Ailments of Man: Edited by Morris Fishbein, M. D. Garden City, N. Y., Garden City Publishing Co., 1945. Pp. 177. Price $1.00.

In this book are discussed many of the ordinary complaints of man. The subjects are written by accredited specialists in their fields. The articles originally appeared in Hygeia. The material is informative and safe in the hands of the laymen. It is a good book to recommend to patients. It stresses the relief and prevention of diseases and yet is so presented as not to encourage the pernicious practice of self medication.

I. L. Robbins, M. D.

Sciatiques et Lombalgies par Hernie Posterieure des Disques Intervertebraux: By D. Petit-Dutaillis and S. De Seze, Paris, Masson et Cie, 1945. Pp. 178.

This monograph from Paris is based on a study of the first 50 cases of sciatica due to ruptured intervertebral disk in which operation was performed by the authors. In general, their conclusions are no different from those of English and Americans on this subject.

The monograph is introduced by a pathogenic and anatomic study and followed by a discussion of the etiology, clinical picture, diagnosis and treatment. Personal statistics punctuate their points in the discussion. The authors have not restricted their discussion to sciatica due to ruptured intervertebral disk but have included compression of the cauda equina as well. They have tried to call attention to the fact that herniated disks are responsible for many neuralgias which were formerly thought to be due to lumbo-sacral vertebral anomalies.

The text is generously supplemented with illustrations and a comprehensive although not complete bibliography is appended. The lack of an index does not interfere with intelligent use of the material as the subject matter and its corresponding pagination are clearly indicated in the table of contents.

Dean H. Echols, M. D.

The Falling Sickness; a History of Epilepsy from the Greeks to the Beginnings of Modern Neurology: By Owsei Temkin. Baltimore, Johns Hopkins Press, 1945. Pp. 380. Price $4.00.

Epilepsy is one of several chronic disorders which has been spoken of in shame and whispers throughout many centuries of its history; however, epilepsy is regarded as a symptom to be controlled rather than a disease entity.

This most thorough narrative review begins with the ancient Greeks who have left us the earliest western writings on epilepsy, and it continues to the point where the historical perspective ends and the present debate begins about the year 1880.

There is no unanimity concerning the range of the concept of epilepsy, and the nature of the disease is as yet obscure. There are many organic diseases which may lead to the same syndrome as is manifested in "genuine" epilepsy and it is very difficult, if not impossible, to differentiate between some mild epileptic attacks and certain mild cases of hysteria. The broader the viewpoint, from which epilepsy is studied, the more the condition seems to lose its identity and enter the domain of convulsive states encompassing many "epilepsies" of different origin.

The history of epilepsy is such a complicated maze of confused and conflicting trends that oversimplification as well as unwarranted inferences must be avoided and interpretation of material made the main task.

No one will expect a solution of strictly medical problems in an historical book. Its aim is to attempt to elucidate the past and thereby try to understand the selling of present issues.

This book is well organized, thoroughly integrated and tremendously informing and its production is a high-level performance.

C. P. MAY, M. D.

PUBLICATIONS RECEIVED

The Commonwealth Fund, New York: The Modern Attack on Tuberculosis, (Revised edition) by Henry D. Chadwick, M. D. and Alton S. Pope, M. D. Medical Services by Government, by Bernard J. Stern, Ph. D.

Grune & Stratton, New York: Current Therapies of Personality Disorders by Bernard Glueck, M. D. Treatment by Ion Transfer, by D. Abramowitsch, M. D. and B. Neoussikine, M. D. Skin Diseases, Nutrition and Metabolism, by Erich Urbach, M. D., F. A. C. A.

The Johns Hopkins Press, Baltimore: Studies on Brazilian Anophelines from the Northeast and Amazon Regions, by O. R. Causey, L. M. Deane and M. P. Deane.

Lea and Febiger, Philadelphia: The Diagnosis and Treatment of Pulmonary Tuberculosis, by Moses J. Stone, M. D. and Paul Dufault, M. D., F. A. C. P.

J. B. Lippincott Company, Philadelphia: A Bibliography of Infantile Paralysis 1789-1944, edited by Morris Fishbein, M. D., compiled by Ludwig Hektoen, M. D. and Ella M. Salmonsen. Surgical Treatment of the Nervous System, edited by Frederic W. Bancroft, A. B., M. D., F. A. C. S. and Cobb Pilcher, M. D., F. A. C. S.

MacMillan Company, New York: Atlas of Surgical Approaches to Bones and Joints, by Toufick Nicola, M. D., F. A. C. S.

Mead Johnson & Company, Indiana: Monograph on Protein Nutrition (Reprinted from J. A. M. A.). Medical Books Published During 1945.

The C. V. Mosby Company, St. Louis: Pneumoperitoneum Treatment, by Andrew Ladislaus Banyai, M. D., F. A. C. P., F. C. C. P.

Philosophical Library, Inc., New York: Narcotics and Drug Addiction, by Erich Hesse.

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THE SURGICAL TREATMENT OF HERNIA*

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The surgical treatment of hernia is an important subject as no present day technic entirely obviates recurrences. The aim of every surgeon should be to make such recurrences as few as possible. Recent improvements in technic have made the present review of the status of the surgical treatment of hernia advisable.

In this paper three aspects of the subjects will be considered.

I. RECURRENT HERNIA AND THE USE OF FASCIAL GRAFTS

The surgical treatment of recurrent hernia, especially in so far as it pertains to groin hernias (indirect inguinal, direct inguinal, and femoral hernias) involves the original procedure as well as the secondary repair. In so far as the original procedure is concerned, I am of the belief that non-absorbable sutures are important in reducing the incidence of recurrence. Furthermore, each step of the original repair should be done carefully as is outlined in Section III of this paper. The rule of Fallis gives a significant lead as to the causation of recurrence of groin hernias. This rule is that hernial recurrences following operations by occasional surgeons establish themselves as indirect recurrences, whereas hernial recurrences following operations in large surgical centers establish themselves as direct recurrences. The interpretation of this rule is that every hernia tends to recur as a direct recurrence, but when the operation is improperly done the sac is often tied off too low and the additional factor of an indirect recurrence exists. The author has personally used free fascial grafts in the repair of eleven recurrent groin hernias and has used interrupted silk sutures with a Cooper's ligament technic in a much larger series of cases. The conclusions, as outlined in a paper by Swenson and Harkins (1943) are that silk is just as good as fascia in most recurrent groin hernias.

II. LARGE VENTRAL AND INCISIONAL HERNIA AND THE USE OF CUTIS GRAFTS

In the repair of large ventral and incisional hernias the important element is to free the fascial ring which, incidentally, is often much smaller than it would first appear to be, and then to suture the edges of this fascial ring to each other. The author has personally never seen an incisional hernia where the fascial edges could not be brought together. Fascial and other types of patches should be used, therefore, only as an adjunct to the major repair and should not be applied to bridge a defect. In performing the major repair free fascial grafts and non-absorbable sutures are, in my opinion, the agents of choice and, of these, non-absorbable sutures are preferable. In most instances, a large hernia will have to be repaired longitudinally, since the original abdominal incisions are in most instances longitudinal. In some cases, however, vertical flaps may be used. Practically always some overlapping of the flaps utilizing mattress sutures by a modified Mayo technic is feasible.

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In eleven cases (1945) I have used cutis grafts as a patch over the larger incisional hernia repair. In all of these instances silk was used to draw the fascial flaps together and the cutis graft was sewed down over the suture line. Cutis is the deep layer of the skin and it is in general prepared by removing the epidermal layer, preferably with a Padgett dermatome, at a depth of about 10/1000 of an inch. The deep layer of the skin is then removed and used for the cutis grafting. The epidermal piece is then sutured down over the donor defect. A piece of cutis about 3x5 inches was used in most of the clinical cases. Microscopic sections revealed that no matter how thick a layer of epidermis was removed, some epidermal elements still remained in the cutis grafts. In dog experiments these lead to sebaceous cyst formation, but in the eleven human patients no difficulty was encountered and so far all of the repairs have remained intact.

III. GROIN HERNIAS AND THE USE OF A COOPER'S LIGAMENT REPAIR

Most groin hernial repairs involve the suturing of the conjoined tendon to Poupart's ligament. The technic I advise involves the suturing of the conjoined tendon to Cooper's ligament. This operation was originated by Lotheissen in 1898 and was shown by McVay (1941) to rest on a sound anatomic basis. This operation was reported by Harkins, Szilagyi, Brush and Williams (1942) as done on a series of 131 groin hernias, and in 1943 by Harkins and Swenson in a larger series of cases. At the present time this series adds up to 362 groin hernias treated by the Cooper's ligament technic and while the follow up is far from complete, I know of only one recurrence. Recently this method has been utilized by Mattson (1946).

Essentially the Cooper's ligament repair of groin hernias involves the following ten steps:

1. *Exposure of the cord and opening of the indirect sac:* In all cases, whether the hernia is indirect inguinal, direct inguinal, or femoral, the indirect sac is opened. In many cases of direct inguinal hernia or femoral hernia, the indirect sac will be normal in size, but can always be found superior and medial to the cord. In cases of complete indirect inguinal hernia of the congenital type, or where the hernial sac is long, it is cut across near the internal ring and separated from the cord. The proximal end is closed by an internal purse string suture as outlined in step 4. while the distal end is left in place. Many surgeons are of the belief that leaving the distal sac in place will cause hydrocele. This has not been my experience and I believe that the hematomas that result from removing the distal sac are far more disastrous than any theoretical occurrences of hydroceles.

2. *Exploration:* Once the indirect sac is opened it is a simple matter to insert the gloved finger and feel Hesselbach's triangle for a direct weakness or obvious direct hernia and to feel the femoral ring. It is indeed surprising how few surgeons will take the extra thirty seconds to perform this exploration and many femoral and direct hernias are overlooked by not so exploring.

3. *Hoguet maneuver:* If a direct sac or weakness is present, or if a femoral hernia is present, it should be transposed laterally to the inferior epigastric vessels by the method of Hoguet (1920). This maneuver is an extremely useful one. In general, no matter how large a direct sac is, it is not opened but is merely transposed. In the case of large direct sacs the transversalis fascia can be infolded with numerous interrupted silk sutures. One advantage of not opening a direct sac is that the danger of opening the bladder is largely obviated.

4. *Internal purse-string closure of the indirect sac:* Such a closure is done with a medium silk suture as high as possible and helps to prevent indirect recurrences.

5. *Plastic on the internal ring:* After the purse-string suture of the indirect sac, it will be noticed that in most cases the internal ring will admit three of four fingers. This should be tightened to give it a

sphincter-like action as advocated by MacGregor (1929, 1945). This tightening is accomplished by suturing the conjoined tendon, the transversalis fascia, and Henle's ligament to each other. The use of Henle's ligament has been especially pointed out by Clark and Hashimoto (1946).

6. *Relaxation of internal oblique:* This is done as directed by Rienhoff (1940) at the junction of the external and internal oblique.

7. *Cooper's ligament sutures:* The conjoined tendon and transversalis fascia are then sutured with heavy silk ligatures to Cooper's ligament. This ligament is an extremely tough thickening on the anterior superior surface of the anterior ramus of the pubis. The first year or so of using this repair I visualized Cooper's ligament by breaking down Hesselbach's triangle. Then for a year or so the sutures were applied blindly, leaving Hesselbach's triangle intact. Recently, however, I have returned again to the principle of direct visualization of Cooper's ligament, having decided that it is easier to apply the sutures under direct vision. The sutures are carried out about one inch to one and one-fourth inches from the anterior pubic spine.

8. *Closure of the external oblique aponeurosis:* In most instances this is done over the cord which is now dropped into the dead space left by the Cooper's ligament sutures.

9. *Closure of Scarpa's fascia.*

10. *Skin closure.*

SUMMARY AND CONCLUSIONS

1. The proper treatment of recurrent hernia involves the choice of a correct original hernial repair as a prophylactic measure and a careful secondary operation as a therapeutic measure. The use of non-absorbable sutures with a Cooper's ligament technic is a satisfactory method for dealing with recurrent groin hernias.

2. The fascial ring should be brought together and closed in ventral and incisional hernias. In certain instances patches of cutis can be grafted over the suture line to give additional support.

3. In a series of 362 groin hernias (indirect inguinal, direct inguinal, and femoral hernias) a Cooper's ligament technic has proved useful.

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PULMONARY HYPERTENSION

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HISTORY

Pulmonary arteriosclerosis was recognized in 1803 by Brisson, but it was thought to be rare until 1815 when Dietrich pointed out its frequency in cases of mitral stenosis. At this time the concept of hypertension of the lesser circulation was a very vague subject and least of all, an entity. From the start of the twentieth century to 1935, secondary cor pulmonale had been attributed to disease of the left side of the heart or to lesions of the parenchyma of the lung, and primary cor pulmonale, to lesions of the vascular bed. Since the work of Brenner in 1935, however, primary pulmonary hypertension has become a disease in which no specific lesion can be found and some writers even doubt if such a condition really exists. On the other hand, secondary pulmonary hypertension, the clinical curiosity of yesterday, has become the "new disease" of today, with its definite "foot-prints" in the lesser circuit being frequently found at the necropsy table at present.

INTRODUCTION

Although the blood pressure within the pulmonary artery cannot be adequately measured, it is realized that such a hypertension can occur and has become a definite entity in the past few years. It has been confused with "pulmonary congestion", "insufficiency of the right heart", and especially with arteriosclerosis of the pulmonary vessels. This concept explains some of the rarer diseases associated with cyanosis such as Ayerza's disease, some of Geisböck's disease, and some of the cases of the Osler-Vaquez syndrome. The recognition of this type of hypertension is of utmost importance from the point of view of circulatory dynamics, in that the compensatory mechanisms involved play such an important role with regard to circulatory disorders. This paper will be devoted to the circumstances under which pulmonary hypertension occurs, the circulatory dynamics involved, the relation to other circulatory states, and to the clinical recognition of such a condition.

CIRCUMSTANCES ASSOCIATED WITH PULMONARY HYPERTENSION

The immediate cause of pulmonary hypertension is unknown; however, certain circumstances are recognized under which an increase in peripheral resistance arises. At necropsy these cases of increased pulmonary peripheral resistance show, with rare exception, an arteriosclerosis of the larger vessels and also an arteriocapillary fibrosis. As you know, such a condition is found in the greater circulation of the arteriosclerotic person with hypertension. The rare exceptions are the very few cases of decrescent arteriosclerosis found in both circuits in extremely old individuals.

Recently it has been recognized that sclerosis of the pulmonary artery and of the aorta are entirely independent of each other, in fact, the simultaneous appearance of such a condition is the exception rather than the rule. It is also known now that pulmonary arteriosclerosis is entirely independent of age or sex. If some toxin, poison, bacterium, or metabolic product caused arteriosclerosis it seems that the process would have to be one affecting both circuits. The experiments in which high cholesterol diets produced arteriosclerosis seem to produce not a true arteriosclerosis but rather a lipoid transformation of the intimal cells. Jores states that to have true arteriosclerosis one must produce changes in the media and there must be hyperplasia of the elastic fibers. These criteria have not been brought out in the experiments mentioned above. Many of the authorities believe the lesion is probably due to stretching of the tissues resulting in replacement fibrosis with eventual hyalin and calcareous deposition, and compensatory hyperplasia of the elastic fibers, muscularis and intima. The scope of this paper limits any further discussion as to etiology of arteriosclerosis. However, definite conditions are associated with an increase in pulmonary peripheral resistance and these will be elucidated now in their order of frequency:

(a) Mitral disease, either stenosis or insufficiency. This is by far the most common.

(b) Permanent emphysema, whether secondary to asthma or to senility.

(c) Widespread obliteration of lung parenchyma such as fibroid tuberculosis, bronchiectasis, pulmonary abscess, interstitial pneumonitis, carcinomatous lymphangitis, widespread pleural adhesions, or intravascular obliteration of pulmonary circuit.

(d) Cardiac disorders leading to prolonged right heart failure, that is, coronary disease, adherent pericardium, "spent" Graves syndrome, or kyphoscoliosis.

(e) Patent ductus arteriosus or other shunts which run from left to right.

As mentioned before, the underlying factor is an increase in pulmonary vascular resistance and even though one cannot measure this resistance, it can be predicted that on a purely mechanical basis the pressure in the pulmonary circuit must be raised in these circumstances. Two groups of mechanisms are concerned: one is on an obstructive basis either within or beyond the pulmonary circuit, and the other is arteriovenous shunts in certain cases of congenital heart disease. Mitral disease or a failing left ventricle with its effects proximately are the only examples of obstruction distal to the pulmonary circuit.

A stenotic mitral valve causes an increased tension within the left auricle which is transmitted backward through the pulmonary vein and thence to the alveolar capillaries. Mitral insufficiency likewise increases the tension within the left auricle, but the incidence, however, cannot be as great as in mitral stenosis, for the resistance offered by the systolic discharge of the left auricle against a tight mitral ring must be much greater than against an incompletely emptied left ventricle. Thus the arteriosclerosis associated with mitral stenosis is much more prominent than that seen with mitral insufficiency.

In emphysema, the increased resistance is the result partly of extensive destruction of the capillary bed and partly by compression and stretching of the intraalveolar arterioles due to the expansion of the alveoli. On the other hand some authorities say that emphysema, *per se* is not an important cause, for although obliteration of the capillaries is extensive, it is extremely unlikely that it ever reaches a degree capable of exhausting the vascular reserve. Also, emphysema is almost always seen in persons who are at an age when myocardial, valvular, coronary and hypertensive heart disease is not at all uncommon. I do not know which "path" to take in respect to this controversy; however, I believe that the increase in pulmonary tension associated with emphysema is exerted equally over the entire vascular circuit and, if true, the amount of vascular reserve would have nothing to do with averting a hypertensive state in the lesser circuit.

Extensive chronic infiltrations of the lung cause an increased peripheral resistance by a destruction of the capillary bed. Pulmonary abscesses with widespread parenchymal reaction or widespread fibroid tuberculosis will greatly encroach upon the vascular bed especially since some arterioles and some of the smaller arteries can be affected by these conditions. Carcinomatous lymphangitis associated with obliterative pulmonary arteritis of small and medium sized arteries secondary to perivascular lymphatic infiltration by carcinomatous cells can very easily destroy enormous areas of the vascular bed. It must not be overlooked that the obliteration of one or two larger radicles of the pulmonary tree will have a more devastating effect than a very widespread encroachment upon the capillary bed alone.

Some writers say that a finding of right sided hypertrophy and dilatation is seen in some patients having sickle-cell anemia. Capillary stasis, owing to distortion and agglutination of erythrocytes, is felt to be the essential factor in producing the obstruction within the pulmonary circuit.

Schistosomiasis (Mansoni) has been shown to cause granulomatous pulmonary lesions within or related to medium and small pulmonary vessels. Many of the small arteries and arterioles showed evidence of intimal thickening and hypertrophy of the media with subsequent encroachment upon the lumen. In many of these patients evidence of right sided hypertrophy and ultimate failure was observed. Most of the organisms ultimately reach the portal system but enough residual infection remains in the lung to cause pulmonary hypertension.

Others state that involvement of the still larger arteries can produce this condition of pulmonary hypertension. In acute cor pulmonale and in thrombosis of the pulmonary artery, if the patient does not die, strain on the right heart can be elicited usually by clinical or electrocardiographic procedures. The authorities are not in accord at all in their beliefs concerning the production of pulmonary hypertension by the last four above conditions mentioned but I give them to you for further study and thought.

The explanation for the increased resistance in the pulmonary circuit following advanced kyphoscoliosis is not entirely clear. It is partly due to compression of the lungs by atelectasis and compensatory emphysema, to kinking of the pulmonary vessels, and to the attacks of repeated bronchitis to which these patients are subject.

Congenital arteriovenous shunts whereby blood from the left heart is thrown into the lesser circulation will naturally increase the tension in the lesser circulation. When a patent ductus arteriosus is present, the higher pressure in the aorta will cause an extra quota of blood to be thrown into the pulmonary circuit. In most cases of septal defect, the reflux of blood is not enough to cause circulatory embarrassment unless another factor exists which increases the arteriovenous shunt from left to right. An example of this is the combination of mitral stenosis with a patulous foramen ovale. Chronic cor pulmonale associated with the tetralogy of Fallot is due to the pulmonary stenosis rather than to the reflux of arterial blood into the lesser circuit. Acquired arteriovenous shunts can be a cause but its rarity permits little or no discussion on the subject. Watjen reports a case of pulmonary arteriosclerosis in a child aged six months, with transposition of the vessels and a septal defect of the ventricles.

These mechanisms fulfill the requirements "laid-down" for the production of pulmonary hypertension, namely, (1) the minute output of the right ventricle; (2) the resistance and capacity changes in the pulmonary circuit; and (3) back pressure resistance produced in the left heart by changes in the systemic circuit.

CHANGES IN CIRCULATORY DYNAMICS RESULTING FROM PULMONARY HYPERTENSION

Due to interference of the passage of oxygen, because of increased thickness and fibrosis of the capillaries of the alveolar wall, and also with the support given by increased venous pressure and stasis, anoxemia is soon to arise. In shunts between the right and left hearts the anoxia is aggravated by the amount of unsaturated blood that is poured into the left heart. The anoxemia results in an increase in the percentage of circulating reduced hemoglobin and this gives rise to a predominant feature of pulmonary hypertension, namely, cyanosis. When failure of the right heart ensues, the cyanosis is enhanced because slowing of the peripheral capillary flow results in an increase of reduced hemoglobin and the increase in venous pressure as mentioned below. The first compensatory result of anoxemia is polycythemia, produced by hyperplasia of the bone marrow. The succeeding compensatory mechanism is an increase in the circulatory blood volume. This is seen only in long standing cases after repeated attacks of failure. The mechanism is allied to the polyuria that follows progressive renal insufficiency.

Increased pressure in the pulmonary circuit may cause an increase of effective venous pressure. The amplitude of ventricular contraction depends primarily upon the so-called initial tension and in the case of the right ventricle this tension is the resistance in the pulmonary circuit. After prolonged increased tension, the ventricular wall will become stretched and a point is reached where a complete contraction is impossible, thus giving way to an increased venous pressure.

Running along concomitantly with the aforesaid mechanism, the following events usually take place. First, hypertrophy and subsequent dilatation of the right ventricle occurs. Second, dilatation of the pulmonary artery with the development of a pulmonary conus visible by x-ray occurs with the eventual development of a pulmonary insufficiency and the development of a Graham-Steell murmur. Occasionally, the dilatation becomes so prominent as to produce dancing shadows at the hilum of the lung. Thirdly, concomitant with the sclerosis of the pulmonary artery there is sclerosis of the chambers of the heart which are subject to the increased tension. This condition affects the left auricle only when the obstructive process is distal to the pulmonary circuit. As a rule the pulmonary veins are also involved by phlebosclerosis except in the case of emphysema. The left ventricle is not affected unless, of course, there is disease of the coronaries or of the greater circulation.

Eventually, when dilatation of the right ventricle becomes extreme, dilatation of the tricuspid ring increases with a subsequent tricuspid insufficiency. This insufficiency is enhanced by sclerosis of the valves and endocardium. This insufficiency causes a reflux of venous blood that brings about congestion in almost all of the viscera in the abdominal cavity. The visceral lesions are probably due to increased venous pressure rather than merely to congestion.

With further strain on the right ventricle dilatation progresses and more and more blood remains in that ventricle at the end of systole, thus causing the right auricle to empty against an ever increasing positive pressure and at this point the increase in venous pressure attains heights that produce obvious effects on the body. As long as the right heart is compensating no outstanding signs and symptoms are present but when a state of chronic failure ensues a host of physiologic and morphologic changes occur.

EFFECTS PRODUCED BY RIGHT SIDED HEART FAILURE

Arteriocapillary fibrosis progressively increases, the alveolar wall becomes greatly thickened and the alveoli decrease in size resulting in a decreased vital capacity. Reversion to the embryonal type of lung takes place. The blood flow is slowed, engorgement occurs, and the already present arteriosclerosis predisposes to infarct formation and consolidation. The result is an ever increasing anoxemia, cyanosis, and polycythemia. The exertional dyspnea seen in the compensated state is now orthopneic and hemoptysis is common. The pleura is affected in that hydrothorax is a common occurrence; however, its mechanism of production is a disputed question at present.

The outflow of blood through the hepatic vein is hindered by the increased pressure in the inferior vena cava. Increased pressure within the portal system is the result and congestion and enlargement of the liver ensues with eventual cardiac cirrhosis and the production of ascites. Some writers believe the cirrhosis to be due to replacement fibrosis induced by pressure atrophy on the central vein of the lobule while Moschcowitz and others believe it is the end result of phlebosclerosis caused by the long standing increased venous pressure—essentially the same process that occurs in the lung, but on the venous side in the liver. Ninty per cent of the cases show impairment of liver function; urobilinogen is usually increased in both the stool and in the urine; hyperbilirubinemia usually parallels the intensity of the right sided failure; and jaundice is seen to occur in the later stages.

At times the venules of the upper thorax become prominent owing to the collateral circulation between the pulmonary and bronchial veins. When pulmonary congestion is severe some of the blood from the right ventricle fails to go through the pulmonary capillaries but instead is shunted into the bronchial veins thence to the azygos and then into the internal mammary veins—the pressure thus being transmitted. to the veins of the pectoral wall.

In the early stages of pulmonary hypertension the spleen in congested and markedly cyanotic. The venous sinuses are dilated and the organ is enlarged but not palpable. When failure ensues the spleen enlarges considerably but due to the concomitant fibrosis and contraction the spleen usually become smaller than normal. This explains why palpable spleens are rare in congestive failure. When the spleen is palpable in congestive failure, cardiac cirrhosis is usually prominent. Similar factors are present here as in the liver when cardiac cirrhosis is present.

The kidneys, associated with uncomplicated pulmonary hypertension, are very little, if any, affected. There may be seen a slight amount of intertubular increase in connective tissue. Obviously, if there is left sided failure, the kidneys will exhibit much pathology. When right sided failure sets in, oliguria with a high specific gravity is first to be seen. The cause of oliguria is obscure but is associated with the shunting of fluid away from the kidney by the development of edema. Proteinuria of a considerable degree is observed and is due to increased permeability of the glomerular capillaries. Casts are usually present and azotemia is common and always associated with oliguria. Excretion tests show a decreased ability of the kidney to excrete dye and this, likewise, is due to the oliguria. As a rule, if the failure is overcome, the kidney will return to normal with very little lasting effect to be seen.

Little is known of the central nervous system in uncomplicated right sided failure. There are no marked anatomic changes noted. Edema is usually absent. There is obvious venous congestion and any rise in pressure in this system must be due to this engorgement. Cerebral disorders observed in right sided failure must be on a functional basis. Fishberg believes that the psychosis so frequently seen is due to dehydration.

Peripheral edema is one of the most prominent signs of right sided failure. At first the edema is latent and indirectly proportional to the amount of diuresis. It is believed that the edema, in the largest part, is due to the increased hydrostatic pressure in the venous end of the capillaries which overcomes the neutralizing effect of the colloidal osmotic pressure of the plasma. When the edema is of long standing, a hypoproteinemia results due to the loss of protein that has escaped into the subcutaneous tissues, contributing to the already existing peripheral edema. Proteinuria may further reduce the blood proteins, and also, an inadequate intake of proteins, or frequent tapping of exudates may lower the blood protein. Increased permeability of the capillaries due to anoxemia may be another factor; however, this is not believed by some investigators because the protein content of the edema fluid is low. It has been shown experimentally that dilatation of the capillaries is the rule in right sided failure and to a certain extent in compensated pulmonary hypertension.

Left sided failure following right sided failure is the exception rather than the rule. However, there are two factors which can explain a secondary left sided failure: (1) a sustained and progressive rise of effective venous pressure must eventually influence the work of the left ventricle. Stroke volume (Starling) is increased and leads inevitably to hypertrophy and dilatation. Now an increased left ventricular discharge is of necessity followed by increased discharge of the right ventricle so that a vicious cycle is set up that takes its toll upon both sides of the heart; (2) the anoxemia must increase the work of the left ventricle which is already overworked by an increased venous return. Such a heart requires more oxygen than normal but the amount is insufficient due to reduction in coronary flow that accompanies hypertrophy and dilatation of the left ventricular muscle. Another vicious cycle is set up.

CLINICAL PICTURE OF PULMONARY HYPERTENSION

There is usually a history of progressive dyspnea out of all proportion to other evidences of congestive heart failure. The dyspnea has as its basis ventilation which immediately suggests its pulmonary origin. Anoxemia occurs with a resulting cyanosis (blue hypertensive) and the patient tends to look older than his age. As the right heart decompensates, a tricuspid insufficiency arises and a markedly elevated venous pressure with an enlarged tender, and possibly pulsating liver is seen. Peripheral edema and ascites become outstanding features. Ascultation reveals an accentuated pulmonic second sound. A Graham-Steell murmur is heard when mitral stenosis is present along with marked "back-pressure effects." If the obstruction is proximal to the alveolar system, the lung fields will be clear. A systolic pulsation may be felt to the left of the sternum and is due to the hypertrophied right ventricle.

The laboratory affords a great deal of confirmatory evidence. X-ray reveals a prominent pulmonary conus, an enlarged right ventricle and auricle, and usually clear pulmonary fields. "Dancing" hilar shadows may be observed by fluoroscopy. If the obstruction is at the mitral valve, the left auricle will be enlarged and the lung fields will be congested.

The venous pressure is increased and the circulation time is increased to a degree that approximately parallels the amount of decompensation.

Finally, the characteristic electrocardiographic pattern indicative of right ventricular hypertrophy and strain lends corroborative evidence. This consists of right axis deviation and inversion of the T wave in leads II, III, and IVF. There may be secondary polycythemia. The criteria for the diagnosis of pulmonary hypertension as set down by the New York Heart Association are:

(1) Radiologic evidence of undue prominence of the pulmonary artery and right ventricle; (2) right axis deviation of the QRS complex in the electrocardiogram; (3) marked dyspnea and cyanosis, and (4) polycythemia.

SUMMARY

Pulmonary hypertension has become a definite entity. This paper has reviewed the more important circumstances under which hypertension of the lesser circuit occurs. The compensatory mechanism and the resulting pathology to such a hypertension has been discussed along with the clinical syndrome observed in most cases. Even though pulmonary tension cannot be measured accurately, let us hope that in the future such an hypertension will be considered and, if possible, combated whenever there occurs a disease in which pulmonary hypertension is a distinct possibility.

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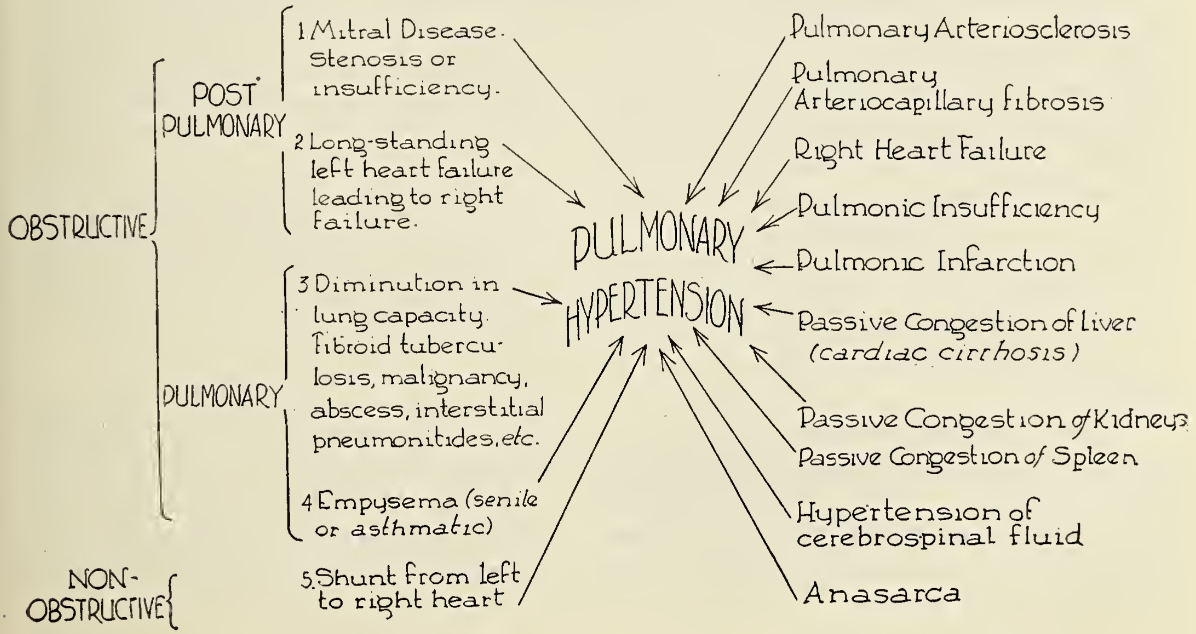
EVOLUTION of PULMONARY HYPERTENSION


OBSTRUCTIVE
POST PULMONARY
1 Mitral Disease. Stenosis or insufficiency.
2 Long-standing left heart failure leading to right failure.
PULMONARY
3 Diminution in lung capacity. Fibroid tuberculosis, malignancy, abscess, interstitial pneumonitides, etc.
4 Empysema (senile or asthmatic)
NON-OBSTRUCTIVE
5 Shunt from left to right heart
PULMONARY HYPERTENSION
Pulmonary Arteriosclerosis
Pulmonary Arteriocapillary fibrosis
Right Heart Failure
Pulmonic Insufficiency
Pulmonic Infarction
Passive Congestion of Liver (cardiac cirrhosis)
Passive Congestion of Kidneys
Passive Congestion of Spleen
Hypertension of cerebrospinal fluid
Anasarca

THE RELATION OF HYPERTENSION TO VASCULAR DISEASE

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INTRODUCTION

Probably one of the most fertile fields for investigation in the past ten years has been the study of hypertension. Interest in this field received enormous impetus by the re-association of the kidney with hypertension, many years after the kidney was first incriminated by Bright, and relegated to a minor role by subsequent workers. This recent work has been productive of many new and interesting discoveries.

The more one reads the literature of hypertension the more he becomes lost in the conflicting maze of theories regarding the changes it produces in the structure and function of tissues. One finds many conflicting reports. For instance, some observers state that arteriosclerosis is one of the causes of high blood pressure; but it must be determined first whether arteriosclerosis is the cause of high blood pressure or hypertension is the cause of arterial degeneration. Moritz and Oldt[67] think that arteriosclerosis is a cause, while Castleman and Smithwick,[18] as well as many others, believe that in an uncomplicated degeneration of the arteries blood pressure does not rise.

In recent years, attempts have been made to subdivide essential hypertension into a large number of different types. Such a separation, in so far as it has been successful, hastened to cast further doubt on the validity of Bright's monogenetic hypothesis. Any one who bases his conclusion on the available clinical evidence alone must necessarily adopt the polygenetic theory. Such a point of view is, however, much too narrow. It is necessary that one surveys all the evidence—experimental as well as clinical.[68]

In this paper, the relation of hypertension to vascular disease will be considered from three primary aspects. First, pathologic variations of the normal physiologic actions which serve to maintain a normal blood pressure. Secondly, pathologic changes in various tissues and organs which are acknowledged to be secondary to hypertension. Last shall be considered the very pertinent, but extremely controversial. problem of the relation of renal arteriolar sclerosis to hypertension.

HISTORY

Hypertension was formerly regarded as always being an expression of either Bright's disease (nephritis) or arteriosclerosis. In 1874, Mahomed described the "prealbuminuric stage of Bright's disease", a term indicating that hypertension may precede frank nephritis but not disassociating the two. Von Basch, 1874, recognized that hypertension may exist in the absence of nephritis and demonstrable arteriosclerosis, and attributed it when present under these circumstances to "latent arteriosclerosis". Allbutt, 1896, differentiated hypertension ("hyperpiesia") from Bright's disease and senile arteriosclerosis. Huchard, 1899, also recognized hypertension in the absence of Bright's disease and, to emphasis its importance in the etiology of arteriosclerosis, called it "presclerosis." "Hypertensive cardiovascular disease" was the designation used by Theodore Janeway in 1913, and it became widely accepted at the time. Frank in 1911 introduced the term "essential hypertension." Volhard and Fahr (1914) and Keith, Wagner and Kernohan (1928), have especially directed attention to cases of essential hypertension which are characterized by kidney involvement and a rapidly progressive, fatal course—"malignant nephrosclerosis" or "malignant hypertension", in contrast to the commoner, "benign" disorder. The importance of cerebral vascular spasm in the production of symptoms was emphasized by Pal (1905) and discussed in detail by Oppenheimer and Fishberg (1928).

DEFINITION

Blood pressure is defined by Wright[117] as the lateral pressure exerted on the walls of the blood vessels by the contained blood. It consists of a systolic phase, which is the maximum pressure in the arteries during contraction of the ventricles, and a diastolic phase, which is a measure of peripheral resistance depending mainly on the tone of the arteries. Hypertension or high blood pressure, according to Wiggers,[112] is a state in which arterial pressure exceeds normal values, taking into account sex, age, race, activity, and temporary psychic states. Generally no doubt as to the presence of hypertension can exist when pressure relations exceed the values 150/100 mg. mercury.

FACTORS WHICH COMBINE TO MAINTAIN THE NORMAL ARTERIAL PRESSURE

Normally there are five physiologic factors operating in the human body as blood pressure determinants: (1) The pumping action of the heart; (2) the peripheral resistance; (3) the quantity of blood in the arterial system; (4) the viscosity of the blood; (5) the elasticity of the arterial walls.[12]

(1). *The Pumping Action of the Heart*—The means by which the cardiac contraction exerts its effect upon the arterial blood pressure is, obviously, through the quantity of blood which it is capable of discharging into the aorta in a unit period of time, that is, upon the output of the heart per minute. When more blood is forced into the already filled arterial system, it cannot escape at once from the system in the same amount as it is thrown into the aorta, so the arterial walls become stretched. The pressure rises until the velocity of flow through the arterioles is great enough to balance again the outflow from the system with the inflow.

(2). *The Peripheral Resistance*—This resides in the arterioles and to a less extent in the capillaries. By far the greater part of the peripheral resistance of the circulatory system is constituted by the arterioles of the abdominal viscera—the so-called splanchnic area. Stimulation of the great splanchnic nerve which innervates the rings of muscle fibres in the walls of these vessels causes their constriction, and consequently a reduction in the outflow from the arterial system. The pressure will continue to rise until inflow and outflow are again balanced. In the absence of compensatory changes in the other factors concerned in the maintenance of the pressure this remains at the higher level so long as the constriction persists. Dilatation of the vessels, that is, reduction in peripheral resistance, will of course be followed by the opposite effect. When the vessels of the splanchnic area are fully dilated they are capable of accommodating almost all the blood in the body; in such an event the blood pressure would fall to zero.

(3). *The Quantity of Blood in the Arterial System*—In any closed system of rigid tubes fluid must fill it to capacity in order that a pressure can be developed within it. The arterial walls are distensible and elastic, and a certain degree of stretching of these must occur before any considerable pressure is created. The arterial system must be actually over-filled and the greater the extent of the over-filling the greater will be the blood pressure. Loss of blood, either of all its elements, as in hemorrhage, or of the fluid portion alone, if not compensated for sufficiently by readjustment of the other factors concerned in blood pressure maintenance, must inevitably result in a fall of pressure. Increasing the total amount of circulating fluid artificially as by the transfusion of blood or of a solution such as saline or gum will elevate the pressure again. Saline solution, however, causes a very evanescent rise, since it soon leaks from the vessels into the surrounding tissues. In animals the blood pressure may be lowered by hemorrhage to a third of its normal value and restored again to its original level by reintroducing into the circulation the blood which has been removed.

(4). *The Viscosity of the Blood*—The greater the viscosity or "thickness" of any liquid the greater is the pressure required to force it along a length of narrow tube in a given time; or if the pressure be constant the longer is the time required for it to traverse the tube. The frictional resistance which is developed between the parts of the liquid itself, that is, the internal friction is greater when the viscosity is high than when it is low. Viscosity depends upon the degree to which the molecules or particles of a fluid cohere to one another. Blood is some five times more viscous than water. With regard to the influence of viscosity upon the blood pressure, it is again a matter of outflow through the arterioles. If the driving force remains constant and the caliber of the vessels is unchanged, then the greater the viscosity the greater will be the frictional resistance developed in this region and the less will be the quantity of fluid that will pass through in a unit of time.

(5). *The Elasticity of the Vessel Walls*—This is concerned mainly with the origin and maintenance of the diastolic pressure and with sustaining the mean pressure at a higher level than would be possible in a rigid system under otherwise identical conditions. The elasticity of arterial tissue does not come into play to any notable extent with a pressure below from 30 to 40 mm. of mercury. Below this level there would be little stretching of the walls of the arteries which would then behave like a system of rigid tubes. At the usual diastolic pressure that exists, however, the walls are stretched and by virtue of their elasticity tend to recoil against the distending force. The flow of blood is pulsatile in the arteries. Beyond the arterioles, that is, in the capillaries and veins the flow is continuous. The conversion of the pulsatile flow to an even one depends upon the existence of a diastolic pressure.[12]

The foregoing facts apply directly to the arterial system. The elasticity of the vascular walls and the peripheral resistance are both essential for the development of the diastolic pressure. As the contents of the ventricle are thrown into the already overfilled system during systole the added pressure which is then exerted upon the vascular walls causes their further distension. After the completion of systole the elastic walls rebound and pressing upon the blood within their embrace force it onwards through the peripheral vessels. In other words, the arterial lumen returns to its previous diameter and the energy that had been stored up during the stretching of the elastic tissue is in this way gradually expended during diastole.[12]

According to Wright,[117] blood pressure varies as a product of the cardiac output and the peripheral resistance. Cardiac output depends upon venous return, the force of the heart, and the rate of the heart. Peripheral resistance, on the other hand, is governed by the viscosity of the blood, the volume of the circulating blood, the velocity of flow, and the size of the lumens of the vessels. The last-mentioned factor is probably the most important in peripheral resistance. The drop in pressure of the blood as it flows through the arterioles is greater than for any other part of the vascular system. Thus, it is thought that these vessels comprise the greatest factor of peripheral resistance. The caliber of the arteriolar lumens is under the control of the vasomotor center by means of the autonomic nervous system. In addition, these vessels are affected directly by metabolites, hydrogen ion concentration, hormones (addenaline and pituitrin), and temperature (body and environmental). The vasomotor center is influenced by impulses from the higher cerebral centers, by carbon dioxide and oxygen concentrations in the blood, by radiation from the respiratory center, but especially by the nerves from the carotid sinus and the aortic arch. These nerves exert a reflex inhibitory action upon the vasoconstrictor center and are probably the most important mechanism for regulation of normal blood pressure, for in addition to their effect on the vasomotor center, they produce effects on the cardiac center in the control of heart rate, and also influence adrenaline secretion, which in turn affects the blood vessels directly. Thus, Heymans[52] considers the regulation of blood pressure as essentially and fundamentally an automatic proprioceptive reflex mechanism.

Besides originating from the carotid sinus and the cardioaortic area, pressosensitive reflexes also arise from the vascular territory of the celiac and mesenteric arteries, and from the territory of the thoracic and pulmonary arteries,[52] as well as from the right auricle and the venae cavae at their entrance into the heart (Bainbridge reflex).

It is to be remembered too that the cerebral vessels and pulmonary vessels are supplied by sympathetic non-medullated vasomotor fibers and that they respond to impulses arriving through these nerves. The coronary circulation is supplied by vagus and sympathetic nerves, the vagus here conveying constrictor fibers and the sympathetic conveying dilator fibers.

PATHOLOGIC VARIATIONS OF THE NORMAL PHYSIOLOGICAL ACTIONS WHICH SERVE TO MAINTAIN A NORMAL BLOOD PRESSURE

(1). *Hypertension Due To Alteration in the Pumping Action of the Heart*—Hypertension due to alteration of this physiologic mechanism is characterized by three clinical findings: (a) increase in systolic pressure; (b) low or normal diastolic pressure, and (c) an obvious increase in pulse pressure.[97]

The predominant cause of increased blood pressure here is the increase in cardiac output, being in turn due to an increase in the minute volume of the heart. The minute volume is determined by two variable factors, that is: (1) the cardiac rate and (2) the amount of venous return to the heart. An increase in cardiac rate will not result in an increase in the minute volume of the heart without a concomitant adequate ventricular filling[12] which presupposes a greater venous return.[97]

Thyrotoxicosis,[95] simple tachycardia due to emotion, or excitement, and arteriovenous aneurysms[32] all result in an increase in venous return to the heart. Thyrotoxicosis and tachycardia exert their effect by speeding up the circulatory rate in the body. An arteriovenous aneurysm increases the cardiac return by mechanical means. An increase in venous return causes an increase in the pressure of the great veins leading to the heart, and, through a reflex mechanism (Bainbridge reflex), the heart is accelerated in rate. Thus, both factors upon which the minute volume of the heart is dependent are increased. Since there is no increase in peripheral resistances, the diastolic pressure remains normal; or, as in the case of thyrotoxicosis, becomes lowered due to peripheral vasodilatation.

Aortic regurgitation and heart block may cause an increase in systolic pressure through a somewhat different mechanism. In these two conditions there is no increase in pulse rate. The increase in minute volume observed is due only to the increased ventricular filling. In aortic regurgitation the incompetent aortic valves allow the reflux of blood from the aorta back into the left ventricle during diastole; the heart muscle fibers are stretched, react more forcibly (Starling's Law of the Heart), and systolic ejection is more complete.[12] Thus an increased diastolic ventricular volume due to a two-way filling (mitral and aortic) plus an increase in efficiency of cardiac muscle results in an increased minute volume and an increase in systolic pressure. The diastolic pressure in this condition is obviously lowered.[97]

Systolic hypertension associated with heart block has a very similar pathogenesis, the chief difference lies in the fact that the increased diastolic ventricular volume is due to the missing of a systolic period rather than to valvular incompetency.[97]

(2). *Hypertension Due to an Increase in the Viscosity of the Blood*—Rarely does this factor play a prominent role in the production of hypertension. Probably the only clinical condition in which the hypertension could be traced to an increased viscosity of the blood is polycythemia vera. Many claim that in the great majority of cases exhibiting hypertension and polycythemia, the hypertension is primary and that the increased red cell count is due to a mild cardiac decompensation. Tinney, Hall and Griffin,[103] however, have fairly well demonstrated that true polycythemia vera may increase the blood pressure. They found that 40 per cent of 163 proved cases with no demonstrable decompensation had an associated hypertension.[97]

With an increase in number of red blood cells there is an increase in viscosity of the blood and an increase in frictional resistance to flow. In the presence of an increased resistance, a constant driving force, and a constant size of the arterioles, there must result a decrease in outflow through the arterioles. Hemodynamical laws demand that, under such conditions, the pressure within the arterial tree proximal to the arterioles must rise in order to force enough blood through the arterioles to equalize the arteriolar inflow and outflow.[12, 97]

This is the same mechanism whereby hypertension is produced when peripheral resistance is increased. The only difference lies in the fact that in the latter, arteriolar outflow is diminished by arteriolar constriction rather than by an increased frictional resistance. Similarly, also, the diastolic pressure is affected to a greater extent than is the systolic.[97]

(3). *Hypertension Due to a Decrease in Elasticity of the Arterial Walls*—Under this heading there will be considered only the larger, more elastic, arteries, such as the aorta and its immediate branches. Arteriolosclerosis, especially of the kidneys, and hypertension will be considered in another section of this paper.

It is a well known fact that, during the normal process of senescence, these vessels undergo atheromatous and calciferous changes; suffer a loss of elastic tissue; and thus lose a large part of their resiliency. As this process progresses, the large arteries concomitantly become less able to "give" with each systolic ejection of blood from the left ventricle. The systolic blood pressure, therefore, rises. In a similar manner these inelastic vessels lose the power of "clamping down" on the smaller diastolic volume of blood. The diastolic pressure falls, remains constant, or rises only slightly. Patients with systolic-diastolic ratios in the nature of 200/90 with little evidence of renal damage or cardiac enlargement, and with peripheral and fundal evidence of extreme arteriosclerosis, should exemplify this type of hypertension.[97]

Cerebral arteriosclerosis may produce an increase in blood pressure through the effect of cerebral ischemia on the vasomotor regulatory centers.

(4). *Hypertension Due to an Increase in Blood Volume*—Blood volume may be increased locally or generally. In either condition there will be an increase in both the systolic and diastolic pressures with a fairly normal pulse pressure.[97]

The localized increase in blood volume is exemplified in the condition of coarctation of the aorta.[14, 85, 86, 88] Here, the mechanical over-filling of the arteries of the upper extremities, head, and neck give rise to an increase in blood pressure in these areas with a decrease in pressure distal to the constriction.[97]

Steele and Cohn[98] report one case of coarctation with a generalized hypertension and suggest that, in some of these cases, the hypertension may be due to renal ischemia. They compare the hypertension in their case to that seen experimentally in constriction of the renal arteries.[43]

A generalized increase in circulatory volume is seen primarily with infusions[12] of hypertonic or protein-containing solutions, and clinically in pituitary and adrenal disorders. Some clinicians have obtained good results in the treatment of patients with essential hypertension by various methods aimed to decrease the blood volume.[97]

(5). *Hypertension Due to an Increase in Peripheral Resistance*—A great deal of experimental work has been done on the mode of production of an increased peripheral resistance, especially in renal diseases and the so-called "essential" hypertension. A survey of the literature in this respect leaves one confused in a maze of theories based upon only a few actual facts at the present time. Suffice it to say that early in all conditions associated with an increased peripheral resistance there is an arteriolar constriction which may be nervous or humoral in origin. In the presence of a constant driving force and an increased peripheral resistance, the pressure in the arterial system must arise in order to maintain the necessary relationship between arteriolar inflow and outflow.[97] Clinically this category is characterized by an increased systolic and diastolic pressure with a variable pulse pressure depending upon the relative degree of change of the two preceding factors.

Increased peripheral resistance associated with ischemia of vital organs, particularly the brain and kidney, may represent primary causes of hypertension. At this point, such conditions in the brain will be considered; those in the kidney will be dealt with later in the paper.

In any condition in which there is an increase in the intracranial pressure, such as in brain tumors[11] or cerebral contusion,[19] certain physiologic mechanisms come into play in order to preserve an adequate cerebral arterial supply. Practically speaking, the cranial cavity contains three fluids; that is, cerebrospinal fluid, venous blood and arterial blood. If for any reason the pressure within the cranium is increased; there is a compensatory decrease in venous blood and cerebrospinal fluid volume. The arterial supply to the brain does not decrease, however, for the pressure within the arterial tree is greater than that within the other two systems. There is, then, obviously an increase in cerebral congestion, since the arterial supply remains constant and the venous outflow decreases. A further increase in intracranial pressure thus takes place. When the intracranial pressure rises above the arterial pressure, a cerebral anemia results, with a concomitant embarrassment to the vital regulatory centers of the brain. In order for life to exist, the arterial pressure must rise. This rise is brought about through a stimulation of the medullary vasomotor centers with a resulting widespread vasoconstriction.[97]

PATHOLOGIC CHANGES IN VARIOUS TISSUES AND ORGANS, ESPECIALLY VASCULAR CHANGES, WHICH ARE ACKNOWLEDGED TO BE SECONDARY TO HYPERTENSION

(1). *Nervous System*—The vast majority of work indicative of possible functional changes in the nervous system has been experimental in nature. The indication seems to be that while there are disturbances in the normal physiologic function of the reflexes and nerves involved in blood pressure control, these disturbances are secondary to the hypertension and not the underlying cause. That a disturbance of the nervous mechanism exists is indicated by the fact that the natural relationship between pulse rate and blood pressure is reversed; rise of blood pressure as seen in hypertension being accompanied by increased pulse rate.[4] Ordinarily, of course, a rise in blood pressure sets up impulses from the carotid sinus and cardioaortic areas, which exert inhibiting influence of the cardiac and vasomotor centers, bringing about slowing of the heart rate and vasodilation, with consequent lowering of the blood pressure. In hypertensives, vasomotor release can be brought about by the application of heat and by novocaine injection of the nerves, so that there results increase in blood flow essentially the same as in normal individuals.[97] This indicates that there is essentially normal vasomotor activity and that the apparent changes are secondary in nature. Thus Pickering[81] states that "normal vasomotor activity is superimposed on an intrinsic vascular hypertonus which is independent of the vasomotor nerves." Further proof of the activity of the vasomotor nervous mechanism is found in the Hines-Brown test. In the light of what is said above, the response in this test peculiar to hypertensives or potential hypertensives may be considered as not a fundamental abnormality of the vasomotor nerves, but a response conditioned and determined by some other underlying factor.

Experimentally, hypertension has been produced by resection of the carotid sinus and aortic nerves.[52, 56] In dogs, the pressure may rise from 130 mm. mercury to 250 or 300 mm. mercury as a consequence of this procedure. This, of course, is due to the release of inhibition of vasoconstrictor and cardioaccelerator centers, the activity of which, under normal conditions, is permanently moderated reflexly by the aortic and carotid sinus nerves. This hypertension persists for a considerable length of time, but it is not permanent. It is prevented or caused to disappear by total excision of the paravertebral ganglionic chains from the stellate ganglia down to the pelvic ganglia.[52] This form of hypertension seems clearly quite different from permanent clinical hypertension, although it is thought by some that there is a type of "neurogenic hypertension" that exists clinically and resembles this experimentally produced hypertension.

In this connection, it might be well to mention that many surgical procedures have been devised wherein various attacks are made upon different portions of the sympathetic nervous system. The effects of these operations on blood pressure are not constant, as reported by Allen and Adson.[5] It is claimed, however, that a relief of symptoms frequently results. That the blood pressure is inconstantly affected further indicates that the functional pathology is not primarily for the nervous system, but for some other factor, involving the nervous system only secondarily, if at all.

Special mention should be made of cerebral circulation and the reaction of pial arteries to increase in blood pressure. Fog[34] found that the usual response of these vessels (pial arterioles) to rises in arterial pressure is by constriction. Sometimes, no change in caliber occurs. This indicates that in these cases also an increase in tonus of the walls of the vessels has taken place. Without such active change in the walls, the sudden rise in pressure would cause passive dilatation. Thus, changes in the intravascular pressure cause changes in the physiologic state (tonus) of the walls of the pial arteries. This tonic regulation persists immediately after bilateral section of the cervical portion of the sympathetic trunk and bilateral section of the vasosensory nerves from the aorta and carotid sinus. The mechanism, then, by which the tonus of these vessels is regulated is not established —it is possibly by intracranial regulation or by local reaction of the smooth muscle of the arterial wall. This type of response to elevation of blood pressure further emphasizes that the cerebral circulation and the pial vessels are no exceptions to the general increase in intravascular pressure. The rise in pressure in the choroidal plexuses with the effect on the cerebrospinal fluid pressure becomes more clearly understood.

(2). *Cardiovascular System*—As stated in the discussion of normal blood pressure regulation, the factors mainly involved are cardiac output and peripheral resistance. Blood volume and the viscosity of the blood are also of some importance, but mainly as factors influencing peripheral resistance. Since hypertension is usually considered as a functional disease of the cardiovascular system, we look for most changes in function and associated structure in this system.

(a) The Heart: The typical heart of essential hypertension shows, pathologically, hypertrophy of the left ventricle with or without associated dilatation.[49, 55, 110] It may be said that a heart weighing over 500 grams is from a case of essential hypertension if other conditions leading to cardiac hypertrophy are absent (valvular disease, glomerulonephritis, hyperthyroidism, adherent pericardium, syphilitic aortitis). These changes, however, are secondary in nature. The heart could be involved primarily only if cardiac output were increased. It has been shown by some workers[101, 110] that the cardiac output is not increased in essential hypertension. In addition to studying cardiac output in hypertensive human beings, Holman and Page[54] investigated the subject in dogs, in which hypertension has been produced by renal artery constrictions. It was found that the bringing on of hypertension did not change cardiac output.

The increased size of the heart and the increased resistance against which it must work in hypertensives bring up the questions as to its efficiency and the functioning of the coronary circulation, since, in the benign type of essential hypertension, death is frequently by heart failure. Wright, Hallaran, and Wiggers[116] have made a study of the "effort index" for hearts of hypertensive subjects. In a comparison of the efficiency of the left ventricle in hypertensives and in normal individuals, they concluded that the left ventricle in hypertension maintains an "efficiency quotient" as good or even better than that of the normal left ventricle, by virtue of a large pulse pressure in spite of an elevated diastolic pressure. Thus, according to Wiggers,[113] physiologically the action of the left ventricle is not impaired as a result of the greater work imposed upon it by severe hypertension. He states further that, in animals, the mechanical efficiency actually increases and the economy with which the mechanical energy is utilized to propel blood is decidedly increased. However, in order to do this, the left ventricle operates continually in the region of cardiac reserve and, if additional distention occurs, for example, because of an added return of blood, (as during exercise) it responds with decreased rather than, as normally, with increased output. This explains the limited capacity for exercise and the development of the "effort syndrome" on exertion.

As regards coronory circulation and blood supply to the heart, it is known[10,113] that coronary inflow increases as does the systolic pressure, and that mechanical adaptations between peripheral coronary resistance and aortic pressure, rather than nervous reflexes or chemical action, are chiefly concerned. It is pointed out by Tollman,[104] however, that, as noted above, the heart undergoes hypertrophy to meet the increased load in hypertension. This hypertrophy increases the mass and diameter of the individual fibers; there is no increase in the number of fibers. The increase in size and diameter of the fibers introduces a metabolic disadvantage, impairing the state of cardiac nutrition. In addition, the frequently concomitant coronary sclerosis may prevent the necessary increase or even maintenance of cardiac circulation. The result is heart failure and a cardiac death.

(b) The Blood: The physical and chemical properties of the blood are important as possibly being involved in the abnormal function of hypertension. Volume and viscosity are factors which, if increased, could be responsible for elevation of the blood pressure. Linder, Lundsgaard, Van Slyke, and Stillman[63] and Austrian[7] have shown that in arterial hypertension both blood volume and blood viscosity are within normal range. In studying dogs with hypertension produced by the Goldblatt technic, Gibson and Robinson[40] found irregular changes in the blood volume. They concluded that there was apparently little relationship between blood volume and hypertension in these dogs.

The most important possible change in the chemical makeup of the blood in hypertension is the presence of substances which may exert a vasoconstrictive influence, the "renal pressor substances"[77]; however, it is beyond the scope of this paper to consider this subject.

(c) The Blood Vessels: From the above data, it is seen that three of the factors (cardiac output, blood volume, and blood viscosity) which might influence blood pressure physiologically, are found to be largely normal in essential hypertension. This leaves only peripheral resistance as the one involved. It seems clear then that there is a fundamental increase in peripheral resistance in hypertension. In fact, Wiggers[113] states that calculations of peripheral resistance by several formulae show that it is increased in various forms of human hypertension. But the magnitude of the increase does not necessarily correspond to the degree to which mean blood pressure is elevated,[39, 60] suggesting that other mechanisms are also concerned.

Peripheral resistance, as mentioned in the discussion of normal blood pressure, is largely determined by the size of the lumina of the arterioles. Pathologically, the essential and universal arterial change in primary hypertension is a muscular hypertrophy, which is proportional in degree to that of the left ventricle.[49] It involves the media chiefly, but the intima also shows reduplication of the internal elastic lamina, and a notable development of new musculo-elastic layers. Kernohan, Anderson and Keith[61] excised small portions of muscle and determined by study of sections that there was an increased thickness of the arterial wall in proportion to the diameter of the lumen of the vessels in hypertension. These changes are almost surely secondary, and represent a physiological response to the increased intravascular tension.

According to Wiggers,[113] organic lesions are not generally widely enough distributed to account for the increased effective resistance. The only plausible view, then, is that the increased peripheral resistance is due to narrowing of the lumina of the vessels by functional contraction or vasospasm. The arterioles and prearterioles are probably most involved, though Wiggers feels that we should not be too dogmatic in assigning the increased resistance entirely to these vessels—some of the resistance may occur in vessels larger than prearterioles.

The work of Prinzmetal and Wilson[82] indicates that peripheral resistance is elevated by means of widespread hypertonus of the arterioles. They studied the blood flow through the limbs of patients with hypertension and found normal values. If only the splanchnic area were responsible for the increased resistance, vasoconstriction here and not in the periphery (muscles) would lead to a greater blood flow through the periphery. These findings are in keeping with the histologic changes in the arterioles of voluntary muscle. Furthermore, blood vessels of hypertensives are capable of dilatation, indicating that the increased peripheral resistance in hypertension is due to generalized vascular hypertonus and not to primary changes in the blood vessels.[97] It has been shown that, with slow injection of dilute epinephrine intravenously into hypertensive persons, the magnitude of the rise in blood pressure (systolic) is the same as in normal individuals.

The role of the larger arteries in the dynamics of hypertension is discussed by Wiggers.[113] He states that diminished elasticity of the aorta and its immediate branches accentuates the elevation of systolic pressure and reduces the diastolic pressure. It accounts also for the large pulse pressure and the rapid diastolic decline of the subclavian pulses in hypertension. An absence of a significant increase in diastolic pressure, far from denoting inconsequential involvement of the peripheral vessels, is probably more often a sign of an extension of vascular changes to the larger vessels. Although sclerosis of larger vessels produces these changes, the suspicion has existed that aortic distensibility is also decreased, even when no pathologic changes are demonstrable. The view that, in such subjects, the aortic distensibility is decreased beyond that common for corresponding ages is supported by considerations arising out of new experiments on circulation models, on the perfused dog's aorta in situ, and by simultaneous records of aortic pressures and diameters of living dogs. These views of Wiggers seem important as additional factors in the pathological physiology of hypertension.

(3) *The Retinal Fields*—The changes here observed ophthalmoscopically are mainly in the retinal arterioles. For that reason, this consideration is taken up immediately following the discussion of the blood vessels. The early vasoconstriction and subsequent structural changes in the arterioles can actually be seen here. The importance of parallelism between the retinal and cerebral vessels is pointed out by Gresser[47] and Salus.[87] According to Wagner[69] the earliest changes observed in the retinal vessels are those of increased arterial tonus, in which the lumen of the arterioles is narrowed but uniform. There is changed or altered relationship between the caliber of the arteriole and the vein as compared with the caliber of those structures in normal subjects. Following this, there is the beginning of recognizable organic change in the arterioles—there is hypertrophy of the tunica media, which causes narrowing of the lumen and gives it an irregular appearance. There is greater reflexion of light from the wall of the arteriole giving it a coppery or steely appearance. Since the veins and arterioles are in the same sheath, the increase in thickness of the arteriolar wall and the increased intra-arterial pressure produce indentation in the more easily compressible veins, the sign designated as arteriovenous compression. Finally, there is what probably represents the beginning of the first recognizable phase of true arteriosclerosis. There is more marked con-

traction and irregularity of the lumens of the arterioles, and exaggeration of the light reflex, associated with increased visibility of the vascular wall, and at times with thickening of the vascular sheaths. In a more advanced stage, in the smallest visible arterioles, there is an appearance of obliteration, often with fine punctate white areas of hyaline or fatty degeneration and punctate hemorrhages. There may be retinitis, characterized by cotton-wool patches or edema of the nerve fibers distal to the points of arteriolar narrowing, occlusion or spasm. Mild edema of the retina and flame-shaped hemorrhages are usually present. There is often edema of the optic disks.[105]

It is pointed out by Salus[87] that the process seems to start in the more peripheral divisions (capillaries and pre-capillaries) and pushes centralward while the disease progresses. Thus, the central artery and its main branches may be still wide while the smaller branches are constricted.

(4) *The Pulmonary System*—The functional disturbances on the pulmonary system in hypertension are entirely secondary. According to Wiggers,[112] a study of heart lung preparations indicates that pressure rises almost proportionately in the pulmonary artery and in the right ventricle and auricle. In man, the blood content of the lungs is increased. These facts are caused by: (a) increased venous return via the coronary system; and (b) increased collateral flow into the pulmonary capillaries via anastomosing bronchial vessels. Thus, vital capacity is reduced as a result of the pulmonary congestion.

Sympathetic non-medullated fibers end (in large numbers) in the media of the pulmonary arterioles and may be fairly safely presumed to be vasomotor in function (Wright[117]). Adrenaline injection and sympathetic stimulation produce vasoconstriction. The pulmonary blood pressure may be raised as much as 40 per cent by sympathetic stimulation, suggesting that the nervous control may be of considerable significance. In any event, whatever factors are responsible for the increased tonus of the splanchnic and peripheral vessels no doubt have an influence on the pulmonary and bronchial vessels with increase pressures here.

THE RELATION OF RENAL ARTERIOLOSCLEROSIS TO PRIMARY HYPERTENSION

Three views of the relationship between renal arteriolosclerosis and primary hypertension have been advocated: (1) that arteriolosclerosis causes hypertension; (2) that hypertension causes arteriolosclerosis; and (3) that the two processes originate independently and only intensify one another when associated together.[10]

The role of the kidneys in the production of hypertension has been under investigation since the time of Bright. That disturbance in kidney function might lead to elevation of the blood pressure has long been known. This formed the basis for the classification of hypertension into renal and non-renal types. Essential hypertension has for many years been considered as the non-renal hypertension, and thus is defined by Fishberg[32] as persistently elevated blood pressure which, on neither clinical nor anatomical grounds, can be considered due to inflammatory or obstructive renal disease. The work of Goldblatt, begun in 1923, has reopened the field and the kidney again is presented as the seat for the functional disturbances seen in hypertension.

It seems pertinent at this point to discuss briefly Goldblatt's famous experiments,[42,43,46] which have consisted in the use of silver clamps whereby the main renal arteries could be constricted and their lumina reduced to any desired caliber. This reduces blood flow through the kidneys. Ischemia of the kidneys is produced and with the ischemia, both systolic and diastolic blood pressures are elevated. When constriction of these arteries is moderate, there is no accompanying disturbance in renal function, detectable by the usual studies of urine and blood. In long standing hypertension of this type, intimal degenerative changes in the retinal arterioles and medial thickening of arterioles in other organs are observed. If constriction is made severe or complete from the beginning, disturbance in renal function usually accompanies the elevated blood pressure and uremia may develop. In some of these experimental animals, there develop widespread fibrinoid and hyaline degeneration and necrosis of the arterioles, with petechiae in some organs, similar in all respects to the lesions observed in the acute malignant phase of essential hypertension in man. Such similarity then to the pictures seen in both benign and malignant essential hypertension in man make this work especially important as indicative of the functional changes in this condition. The view that the kidney may play a primary part in the development of essential hypertension is reenforced.

In order to attempt to clarify the controversial subject in question, it will probably be well to consider briefly the latest experimental studies of several of the outstanding workers interested in the relation of renal arteriolosclerosis to hypertension.

Bell[10] attacks the problem in this manner: If it is true that renal arteriolosclerosis causes hypertension, then all cases of hypertension should show arteriolosclerosis, and one might also expect that all cases of arteriolosclerosis would show hypertension. As a means of studying this problem, microscopic studies of the kidneys were made from 267 nonhypertensive individuals, all over 40 years of age. There were no hypertensive symptoms, the hearts were of normal size, and the systolic blood pressure was always below 140 mm. mercury. All cases of diabetes were excluded. In this control group 17.8 per cent showed renal arteriolosclerosis of grade 1 or higher, and 80 per cent were grade 1. When the cases were arranged with respect to age, it was found that arteriolosclerosis (grade 1 or higher) occurred in about 9 per cent of those between 40 and 60 years old, and in about 30 per cent of those over 70 years old. It is clear, therefore, that renal arteriosclerosis is an age change and that it occurs in a great many elderly individuals who do not have hypertension. If arteriolosclerosis is the only cause of hypertension, it is not clear why these individuals with arteriolosclerosis did not have an elevated blood pressure. It cannot be argued that hypertension was the cause of the arteriolosclerosis in these controls.

In autopsy studies, renal arteriolosclerosis has been found to be about four times as frequent in hypertensive persons as in nonhypertensive controls, but a great many typical cases, especially of the myocardial and coronary forms, do not show arteriolosclerosis. Those who insist that hypertension is always caused by renal vascular disease maintain that these cases without arteriolosclerosis have lesions in larger arteries sufficient to cause renal ischemia. However, sclerosis of large and small renal arteries is present in the majority of nonhypertensive elderly persons, and some of the hypertensive subjects have no disease even of these larger vessels.

Bell has studied the kidneys microscopically in about 600 persons with diabetes. It is well known that generalized arteriosclerosis is much more severe in elderly persons with diabetes than in persons without diabetes of corresponding age. Renal arteriolosclerosis is also very prominent in persons with diabetes and occurs in nearly half of those with small hearts and systolic pressures below 140 mm. mercury. Bell has found the incidence of arteriolosclerosis in diabetes to be greater with increasingly higher levels of systolic blood pressure.

It is obvious that there is a causal connection between hypertension and renal arteriolosclerosis in diabetes, but it is noteworthy that either condition may exist without the other. Age is a factor of great influence, since renal arteriolosclerosis was found in 31.8 per cent of persons with diabetes under 50 years of age and in 80 per cent of those over 50 years old.

The available data from Bell's studies indicate strongly that renal arteriolosclerosis is primarily an age change in the arteries and that when it develops to a sufficient degree, it usually brings about or intensifies hypertension, but in a great many instances it does not cause hypertension. Typical clinical hypertension may develop in the absence of renal arteriolosclerosis. It appears, therefore, that renal arteriolosclerosis and hypertension may originate independently, but when the vascular lesion becomes severe, it usually causes hypertension.

The evidence that hypertension causes renal arteriolosclerosis is not convincing. One argument against this view is the frequent absence of arteriolosclerosis in hypertensive disease of long duration. In chronic glomerulonephritis with hypertension for many years, true renal arteriolosclerosis is not often seen. The sclerotic arteries in this disease are usually examples of disuse atrophy. It is clear, however, that hypertension causes lesions in the retinal arteries, since these are not present at first and appear only after the blood pressure has reached high levels. It is easily possible that hypertension hastens the process of sclerosis elsewhere in the vascular system.

Moritz and Oldt[67] have recently made an extensive study of the arteriolar changes. They found that the objective examination of arterioles in all parts of the body of 100 control cases and 100 cases of chronic hypertension disclosed only one situation in which the presence of arteriolar sclerosis was almost invariably associated with hypertension and where the absence of arteriolar sclerosis almost invariably betokened an absence of high blood pressure. This was in the kidneys. Renal arteriolar sclerosis was present in 109 of the 200 cases studied, and 97 of these 109 proved to be cases of chronic hypertension. No comparable correlation could be found in any other organ or tissue.

They felt that these facts, together with the information gained from a study of the histologic characteristics of arteriolar disease in hypertensive and nonhypertensive individuals, supported the conclusion that renal arteriolar sclerosis is the most common cause of chronic hypertension. This conclusion is in accord with the recent demonstration by Goldblatt that chronic hypertension is regularly produced in dogs and monkeys by reducing the blood flow through the kidneys (renal ischemia). They believe that the effect of the renal arteriolar sclerosis in human hypertension appears to be the functional analogue of the renal arterial clamp in experimental hypertension. In both instances, hypertension does not necessarily lead to a sufficient degree of ischemia to impair renal function measurably.

They concluded that the only significant site of arteriolar sclerosis so far as the causation of hypertension is concerned, is the kidney.

Castleman and Smithwick[18] were afforded an unique opportunity to examine grossly the kidneys of hypertensive patients and to take biopsies therefrom, by the introduction of the surgical treatment of hypertension by dorso lumbar sympathectomy. From 1941-43, these workers collected renal biopsies from more than 100 hypertensive patients.

The 100 patients in this series ranged from 18 to 56 years of age, the average being 39. There were 43 males and 57 females. Their symptoms included headache, fatigue and occasionally visual disturbances. These had been present from one month to 15 years—average six years. The average systolic pressure was 210 and the average diastolic 130. Renal function determined by the PSP test showed that 60 per cent had normal function, that 20 per cent had a moderate reduction in excretion of the dye and that 20 per cent had a decided reduction.

Each biopsy was graded according to the severity of its vascular disease without knowledge of any clinical data about the patient, and it was found that there were enough differences in the degree of vascular disease to classify the group into five grades: grades 0, 1, 2, 3 and 4. The biopsies showed that a very few patients, usually the older ones, had an occasional focal scar with vascular disease in it. These scars are seen so often in nonhypertensive patients that it was felt that they should not influence the grading of the generalized vascular condition. This policy was also employed by Moritz and Oldt.

The most striking result of this work is the finding of such a high percentage of renal biopsies in which there is no or only minimal vascular disease. Seven per cent were graded 0, 21 per cent grade 1 and 25 per cent grade 2, that is, 28 per cent and possibly 53 per cent of the cases showed renal vascular systems so slightly damaged that it seemed unlikely that the blood flow could have been embarrassed sufficiently to be the one factor responsible for the hypertension. A valid objection might be raised that biopsy as small as the ones removed in this series is not an adequate sample of the whole kidney. It is believed, however, by Castleman and Smithwick that since great care was taken to select a representative region for biopsy and since similar morphologic findings were present in both kidneys in those 25 cases in which bilateral biopsies were taken, each biopsy is a fair sample of the renal tissue in the respective case.

When one considers that seven cases in this series, some with long standing hypertension, had no renal vascular disease at all, grade 0, one is led to believe that the hypertensive state antedates the vascular disease. It can further be stated that, in many of the 25 cases presenting grade two renal vascular disease in which the changes did not appear severe enough to have diminished the vascular bed to cause hypertension, the hypertension may also have antedated the vascular disease. This is in keeping with Cox and Docks' recent perfusion experiments on kidneys postmorten in which they found that "most kidneys from patients with hypertension without uremia have vascular beds in the normal range."

SUMMARY

In this paper, material under the following headings has been considered:

1. Factors which combine to maintain the normal arterial pressure.

2. Pathologic variations of the normal physiologic action which serve to maintain a normal blood pressure.

A. Hypertension due to alteration in the pumping action of the heart—thyrotoxicosis, tachycardia, aortic regurgitation, and heart block.

B. Hypertension due to an increase in the viscosity of the blood—polycythemia vera.

C. Hypertension due to a decrease in elasticity of the arterial walls—arteriosclerosis.

D. Hypertension due to an increase in blood volume—coarctation of the aorta.

E. Hypertension due to an increase in peripheral resistance — associated with ischemia of the brain.

3. Pathologic changes in various tissues and organs, especially vascular changes, which are acknowledged to be secondary to hypertension.

A. Nervous system.

B. Cardiovascular system.

(1) The heart.

(2) The blood.

(3) The blood vessels.

C. The retinal fields.

D. The pulmonary system.

4. The relation of renal arteriolosclerosis to primary hypertension.

A. Brief historical review.

B. Recent experimental work.

CONCLUSIONS

1. Certain pathologic variations of the factors controlling normal blood pressure can produce hypertension.

2. The nervous system, cardiovascular system, retinal fields and pulmonary system frequently show evidence of damage secondary to hypertension.

3. A state of hypertension not infrequently exists in man in which evidence of renal vascular disease is either absent or is insufficient to explain the elevated blood pressure. The implication is that in many cases some other functional factor or factors exist which are primarily responsible for the hypertensive state and which precede the appearance of renal vascular disease. It is certainly very probable that the vascular disease, once established, aggravates the already present hypertension, which in turn accelerates the arteriosclerosis—vicious circle. This is the stage in which the kidney is found postmortem and on which the theory of the primary

vascular origin of essential hypertension is based. Observations of the kidney biopsies in the earlier stages of hypertension have given the missing link to help substantiate the theory that the hypertensive state precedes the renal vascular disease in many cases.

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GASTRIC POLYPOSIS

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INTRODUCTION

Gastric polyposis is indeed a rare condition; however, its rarity does not preclude a knowledge of the subject. Gastric polyposis should constantly be considered in the differential diagnosis of gastric carcinoma, gastric ulcer, other benign tumors of the stomach, and possibly should be watched for in patients who have pernicious anemia. Therefore, it is the purpose of this paper to acquaint the reader with the theories of etiology, the histology, signs and symptoms, diagnosis, therapy, and prognosis of gastric polyposis so that he will be better able to cope with the vague gastric complaints with which he will be faced upon entering the practice of medicine.

DEFINITION

Galen originated the term polyp for describing pedunculated tumors of the nasal mucosa, it refers not to a structural property but merely to the form which may be assumed by any growth. Gastric polyposis then is the occurrence of multiple benign polypoid growths of the gastric mucous membrane involving diffusely the gastric mucosa.

HISTORY

The first gastric polyp is said to have been described by Amatus Lusitanus (1557). However, the diagnosis is not clear, and no autopsy was done. In 1761 Morgagni described a gastric polyp in a woman fifty years old who had died of dropsy. Cruveilheir (1835) wrote of the possibility of malignant change in polyps; he also wrote of the possibility of their obstructing the pylorus. Quain in 1857 reported a case of a nineteen year old girl who vomited a polyp with its pedicle. Rokitansky (1861) described gastric polypi in his textbook, and in 1864 Ebstein gave a detailed review of the literature of that time. In 1888 Menetrier gave his classical paper on gastric polyadenoma. In 1908 Wegele made the diagnosis of gastric polypi when he found a small polyp in the lumen of a stomach tube he had used in examining the patient. The diagnosis was made in a similar manner by Meyer and Chosrojeff in 1909. In 1911 Heinz made the diagnosis when he found a piece of adenomatous tissue in stomach washings; this was confirmed at operation. In 1912 the first roentgen film of gastric polyps was taken. In 1913 a case of polyadenoma associated with carcinoma was reported. During this same year Meulengracht used the term gastritis polyposa and brought careful histologic evidence to support the view that polyps were the result of chronic gastritis. In 1921 Eusterman and Senty presented a paper on the radiologic findings. The introduction of the gastroscope, at this time, as an aid to roentgen diagnosis, was a tremendous step forward and its use becomes constantly more important.

INCIDENCE

The relative frequency of this condition is difficult to estimate, since there are no complete statistics available, and since much of the early data on this subject are not reliable. Also because of the lack of symptoms many cases are overlooked. There were only 84 cases reported in the literature from 1820-1926, and only 41 cases reported from 1926-1941. This is probably not due to an actual increase of the condition itself, but rather to an increase in the diagnostic acumen of the modern physician, and his willingness to report.

Two-thirds of the cases of gastric polyposis are discovered in the fourth and fifth decades; however, cases have been reported in individuals as young as 23 and as old as 80. The condition is more frequent in men than women, the ratio being approximately three to two.

ETIOLOGY

There are two recognized theories regarding the etiology of gastric polyposis, one is the congenital, the other the inflammatory. In the former it is believed that the tumors are of congenital origin, being true neoplastic growths arising from cells in the gastric mucosa which have retained their embryonic properties which are usually lost, especially that of proliferation. The upholders of this theory ascribe the inflammatory reaction usually found around such tumors to mechanical injury. In the latter theory it is believed that the growths arise from a chronic inflammatory gastritis, and Konjetzny claims to have found all the stages from hypertrophic gastritis to adenoma and finally to carcinoma.

Evidence for the congenital origin of gastric polyposis may be summed up as follows: The epithelial structure of the polypi is often irregular and atypical suggesting new growth, and the undifferentiated cuboidal cells are suggestive of an embryonic structure. The occurrence of the disorder in twins, three, four, and six siblings, and in a mother and son seems to support this contention. Occasionally a case has been reported in the newborn, also the hereditary factor is observed in diffuse alimentary polyposis of children, which may involve the stomach, duodenum, and colon. Weschselmann noted outspoken familial predisposition in 50 to 60 per cent of the cases in which the whole gastrointestinal tract was involved; however, Brunn and Pearl in a series of 135 cases found not one case in which there was an unquestionable positive hereditary factor. Another point in favor of this theory is the fact that the gastric polypi are indistinguishable anatomically from the intestinal variety. Still other points are the facts that evidence of inflammation is often wanting, that there is a clear margin between normal and abnormal tissue, that gastritis is common and polyps are rare.

Along experimental lines, proof of the congenital theory is given by Stewart who has reported the occurrence of diffuse lesions, characterized by adenomatous, hypertrophic, and hyperplastic overgrowth of the glandular rugae, in the pyloric chamber of almost everyone of his strain I mice. This is definitely inherited as a recessive character.

In favor of the inflammatory theory are the following: Gastritis though usually followed by atrophy, may cause hyperplasia of both epithelial and connective tissue; in certain areas of polyposis evidences of a preceding gastritis may be found; however, this evidence is usually wanting. Though polypi may be found in infancy, those of the most clinical importance usually seem to evolve in later life.

Experimentally, in animals, polypi have been produced by chronic irritation. Japanese workers have produced polypoid lesions by the injection of coal tar into the gastric mucosa of rats. Tsukiaskas found marked epithelial hypertrophy in the stomachs of two monkeys, probably produced by the irritation from ascaris. Fibiger fed a certain nematode to rats, and produced in the stomach mucosa wide-spread polypoid growths, the initial stages of which were epithelial hyperplasia and inflammation; he was able to follow the lesion to malignant degeneration and unquestionable metastasis. Fibiger believed that these gastric changes were produced by toxins liberated by the nematode. Wassink confirmed this work. Otto, reports a case, in man, in which a thick piece of wood was swallowed, causing marked gastritic symptoms for a year, and finally resulting in death. At necropsy a large gastric adenoma was found, which on section showed wood in its center. Zabel found large numbers of megastoma intestinale in the tufts of a large gastric papilloma.

Some doubt is cast upon the theory of inflammation by the statistics of Lange who found no polypi in 46 cases of chronic gastritis but only slight hyperplastic phenomena. Also Perinin in 50 cases of chronic gastritis, in tuberculous subjects, found no evidence of polypi.

There are other lesser theories of origin of gastric polyposis. Some authorities believe the cause to be a primary degeneration of the glandular epithelium, not congenital, and the inflammatory changes to be secondary; and that the polypoid form is the result of mechanical pull by the action of the stomach on a growth already present. There are also those who believe the growths to be due to such causes as chronic alcoholism, arteriosclerosis of the gastric vessels, syphilis, amyloid degeneration, and dietetic errors, but these theories are not generally supported.

Brunn and Pearl are of the opinion that there are two types of epithelial overgrowth, an hyperplasia secondary to inflammation, and an hypertrophy congenital in origin. They found on studying sections taken from a large number of cases which conformed more to the congenital type, that the gastric epithelium assumed the dominant role, the connective tissue being relatively passive, and that evidence of chronic inflammatory changes is distinctly secondary.

Some authorities have combined the two main theories of origin concluding that certain cells are congenitally disposed to overgrowth and that inflammatory stimuli may be precipitating factors. This suggestion does something to account for the discrepancy between the frequency of gastritis and the rarity of polyps.

At the present time a great deal of interest is being paid to the relationship between gastritis, adenoma, and carcinoma, as first reported by Konjetzny. The high incidence of achlorhydria in cases of gastric polyposis indicates that the condition is associated with widespread changes in the gastric epithelium. In over 60 per cent of the cases of gastric carcinoma similar types of secretion have been reported. Schlindler in the gastroscopic examination of 2167 patients discovered 36 cases of multiple and single adenoma, 26 of which had a definite gastritis; of this number 20 were atrophic, two superficial, and four hypertrophic in type. Conversely 0.83 per cent of 1,814 patients without atrophic gastritis had visible adenomas, whereas 6 per cent of 353 patients with atrophic gastritis had visible adenomas, whereas 6 per cent of 353 patients with atrophic gastritis had adenomas.

The high incidence of gastric achlorhydria found in cases of gastric polyposis has brought about the question of the relationship between this condition and pernicious anemia. Schindler found gastric adenoma in six of 43 patients suffering from pernicious anemia, this being a much greater incidence than that found in patients not having pernicious anemia. Similarly, Rhoades found that gastric polyposis was more commonly found in persons dead of pernicious anemia, than those dead of other causes. In a postmortem study of patients dead of pernicious anemia Brown found chronic gastritis commonly present. She noted in 151 autopsies on such patients that the incidence of benign gastric tumors was 8 per cent, whereas the incidence in 18,200 patients without pernicious anemia was 0.03 per cent.

PATHOLOGY

The first classification of gastric polyposis was made by Menetrier in 1888. He described two types—polyadenome polypeaux, and polyadenome en nappe. In the former there are many discrete polyps, varying from one to several hundred in number, and scattered over the gastric mucosa. They are usually pedunculated, and may bear cysts. In the latter type the hypertrophy forms a well demarcated plaque, not cystic or pedunculated. It is composed of closely placed folds of hypertrophic mucous membrane resembling, in many instances, the convolutions of the brain. Menetrier believed the polypeaux variety to be due to hypertrophic changes involving the ducts and necks of the glands, with the stalk and tumor pattern formed by the connective tissue elements derived from the submucosa; the en nappe type is thought to be more purely epithelial growth which involves the fundus of the glands.

Pearl and Brunn are of the opinion that the polyadenome polypeaux conforms to the type which is considered as congenital, whereas the polyadenoma en nappe corresponds to the description of a localized hypertropic gastritis. Menetrier, however, was of the opinion that both types were due to chronic irritation.

Thus, it is generally recognized that there are two types of gastric polyps—the congenital, neoplastic adenoma, and the acquired hyperplasias due to chronic irritation. It is frequently impossible to differentiate these two types of polyps on gross examination. Grossly the congenital type are soft polyps of various sizes. They are usually pedunculated but may be sessile, and they usually have a tufted or tree-like appearance. They are freely movable on the submucosa and each tumor is separated by an area of normal mucosa. Inflammatory processes are slight or absent. The mucous membrane away from the polyps may be normal or show an atrophic gastritis with reduction in gland tissue and achlorhydria. On section the connective tissue, which originates from the submucosa, seems to invaginate itself into the growth as a central fibrous tissue core. This connective tissue pushes the muscularis mucosa before it and the latter splits into strands as the connective tissue enters the base of the polyp. The connective tissue may be edematous containing thick or thin collagenous bundles. Resting upon the central core is the glandular epithelium which is arranged in a disorderly fashion into glands which show a considerable variation from the normal. Many are elongated, tortuous and dilated; some have been changed into small cysts containing old secretory material. At times the epithelial lining shows proliferation in the form of atypical papillae. Superficial ulceration, necrosis, and inflammatory infiltration often occur. The epithelial cells may be of the undifferentiated, tall hyperchromatic type, or of the type resembling the cells of the small intestine. There may be a mixture of goblet cells, Paneth cells, and the fuchsinophil cells of Lubarsch, with variations from one to another, even in the same gland.

The hyperplastic polypi are also of various forms. The gastric folds or rugae are enlarged, looking pressed together like the convolutions of the brain. The color varies according to the intensity of the process. In the acute and subacute state it is red and injected and may show hemorrhagic and purulent specks, or in more severe cases erosion, ulceration or necrosis may be seen. In the larger chronic edematous swellings the color is paler with mucus lying on or between the folds. The inflammatory hyperplasias have defective margins which merge with the normal mucosa. Their edges have a slow rise to a hilly slope, and they are flatter and more expansive on account of the inflammatory reaction. One does not receive the impression of an independent suddenly arising, isolated tumor. They are firm and rest immovable on the submucosa. Inflammatory polyps are rarely differentiated into head and stalk. On section the submucosa is seen to take no part in the growth as a connective tissue core, and the neighboring mucosa shows definite signs of chronic inflammation. The enlargement which forms the polyp is composed purely of mucosal proliferation of which the epithelial and fibrous components play a part. The epithelial cells lining the surface and those lining the crypts, including even the chief and parietal cells of the fundal mucosa, are replaced in areas large and small by intestinal type goblet cells and Paneth cells, this being a metaplasia or an acquired heteroplasia. There is also a relative increase in gland tissue which accounts for the over secretion of mucus, but the glands do not become so irregular and cystic as they do with an adenoma. The crypts contain a purulent mixture of mucus and leukocytes, which also lies on the surface. These hyperplastic areas are not sharply delineated, but merge imperceptibly into the adjacent mucosa.

In some cases both the neoplastic and hyperplastic types may appear in the same specimen. At times the microscopic picture does not fit readily into the classification given, making it difficult or even impossible, to place the abnormality in a clear cut neoplastic or hyperplastic category.

MALIGNANT DEGENERATION

Although gastric polyposis is essentially a benign condition it is potentially capable of undergoing malignant degeneration. As stated succinctly by Kirklin and Broders, "Although polyps are at least primarily benign, they hold their benignant status precariously." Ewing has expressed the belief that all bukly gastric carcinomas may arise in benign polyps. It is the general consensus that the development of malignancy is precipitated by the following factors; the trauma caused by gastric peristalsis and the passage of coarse articles of food, the adenomatous nature of the benign lesions, and the chronic inflammation which may be pressent.

Malignant degeneration in gastric polyposis is similar to that occurring in epithelial tissue elsewhere. The usual changes are as follows; the epithelium becomes multi-layered, more undifferentiated, and exhibits polymorphism. The epithelium grows in an irregular fashion and mitotic figures become frequent. Finally the epithelium breaks through the muscularis mucosa and infiltrates the submucosa. It is only at this stage that one can be sure that the growth has assumed a malignant character.

The frequency of malignant change in gastric polyposis has been variously reported from 4.9 per cent to 41.2 per cent. The 12 per cent quoted by Brunn and Pearl is perhaps a figure which can generally be accepted. Regardless of the statistics reported, however, malignant degeneration should be suspected in all cases of gastric polyposis and the condition should be treated with this thought in mind.

SIGNS AND SYMPTOMS

The clinical picture presented by gastric polyposis is in no way characteristic. The signs and symptoms are extremely variable in both degree and duration. They vary according to the size, type, and location of the lesions. The condition may remain asymptomatic throughout life and only be found accidentally at necropsy. The duration of symptoms has been reported up to twenty-five years. There may be frequent remissions of varying duration, the longest reported being nine years. The returning symptoms are usually of a much greater severity than the original ones. The patient may subjectively experience vague abdominal distress, such as a dragging or a fullness in the epigastrium. He may have the sensation of slight pressure in the epigastrium, or that of severe pain. This pain has no distinct relationship to the taking of food, as the pain has been reported to come on before, during, and after meals. Some patients experience a marked anorexia, but the majority of patients have no changes in appetite. This is in direct contrast to those individuals having gastric carcinoma. The patient may complain of frequent eructations, nausea, and vomiting. They frequently notice that the vomitus is peculiar in that it resembles egg white very closely. This is due to the excessive mucus which the vomitus contains. Frequently the patient will notice gross blood in the vomitus, and upon occasion a small particle of a polyp or a whole polyp may be found. Occasionally the patient will notice that he has passed a polyp in the stool. The tearing loose of a polyp from its base is associated with severe pain, and a variable degree of hemorrhage, which may or may not give rise to hematemesis and melena. Depending on the amount of hemorrhage will be the degree of anemia and related weakness from which the patient will suffer. A polyp with a long pedicle may prolapse into the pylorus causing complete or partial obstruction. It may prolapse completely through the pylorus into the duodenum giving rise to a gastrogenous diarrhea. On physical examination one finds little or nothing. One may notice evidences of an anemic pallor, and recent weight loss. Examination of the abdomen may reveal slight generalized rigidity, and mild to moderate tenderness in the epigastric region. In a very small percentage of the cases a palpable mass may be felt. This occurs most commonly in patients in whom the growth is large and located in the pyloric region.

The laboratory examination is extremely important in patients suspected of having this condition. The laboratory findings upon the examination of the gastric secretions are quiet significant. A large percentage of the patients (nine out of ten) will have an achlorhydria. One may also find associated with this that the gastric secretions are without digestive property and that they contain increased amounts of lactic acid. The egg white consistency of the secretions is frequently noted. Occasionally one may find particles of a polyp or a whole polyp in the gastric washings. Examination of the blood may reveal a secondary anemia depending on the amount of hemorrhage which has taken place. One may occasionally find a blood picture resembling that of pernicious anemia. Fecal specimens may be grossly bloody, and occasionally a polyp may be passed in the stool.

ROENTGEN AND GASTROSCOPIC EXAMINATION

One might think that the roentgen findings in gastric polyposis are pathognomonic of this condition, but this is not true. There are many times when even the most expert roentgenologists are hard pressed in differentiating gastric polyposis from gastric carcinoma. Gastric polyposis may present a roentgenographic picture so similar to gastric carcinoma that differentiation is impossible. However, the combined use of the roentgen ray and the gastroscope greatly increases the number of correct diagnoses, and a diagnosis of gastric polyposis should not be made until both roentgen and gastroscopic examinations have been done.

Before gastroscopic and, especially, roentgen examinations are carried out the stomach should be empty, for foreign bodies, such as food, excess mucus, and hair balls, may greatly confuse the picture. Thus, the patient should take no food or fluid after his evening meal preceding the morning of examination.

In the roentgen examination the barium mixture should be of such viscosity that it will coat the furrows of the tongue. The mixture should be given in small quantities —three to four teaspoonfuls at a time. The patient, who has been properly prepared, is screened for opacities. During the screening examination it may be noticed that peristalsis is unimpaired and that the stomach walls seem flexible. Overactive peristalsis will suggest obstruction. The tumor may be freely movable to gentle pressure; and, occasionally, a 'crater' appearance may be seen on a tumor, which appearance at the time of operation is proved to be an ulceration. The emptying time is usually normal. During this screening process 5 c.c. of fresh hydrogen peroxide may be given with three to four tablespoonfuls of barium—bubbles indicating blood, mucus, or other loose organic material.

After the screening process the patient is placed in the reclining position on a Potter-Bucky diaphragm, and trained to hold his breath on both inspiration and expiration. This is advantageous in that pressure can be made on parts otherwise covered by the ribs. Compression is used when the opaque media tends to pool in the stomach; otherwise, breathing movements and alterations in posture do what is needed. Films are taken with the patient sitting up, in the supine and prone positions, and also in the right and left oblique positions if desired. A funnel should be used to confine the radiation to the area which is being inspected.

Examination of the films will show the lesions, in the majority of instances, to be located on the greater curvature in the lower third of the stomach. The polyps project into the gastric lumen and produce ragged indentations in the barium shadow. These resemble mottled, fingerprint filling defects.

On gastroscopic examination, which is

excellent for the study of the gastric mucosa, one may see small reddened prominences separted by valleys of grayish mucosa. The prominences may contain many yellow ulcerations, and their surfaces may be irregular and nodular. The remaining gastric mucosa often appears normal.

It is important that the reader fully realizes that the differentiation of benign from malignant gastric tumors cannot be made on roentgen or gastroscopic examinations. Some roentgenologists claim that the defect in malignancy is annular, involving both curvatures, and, thus, can be differentiated from a benign lesion. Still others claim that infiltration of gastric musculature can be seen on the roentgenogram. Some gastroscopists claim to have the ability to make the differentiation between benign and malignant conditions by characteristics which are visible through the instrument, but not apparent in excised specimen or at autopsy. Until these premises can be definitely upheld by further study, all cases of gastric polyposis should be considered as malignant.

DIAGNOSIS

As has been seen from the above, the clinical picture alone is not characteristic enough to warrant a definite diagnosis of gastric polyposis. However, the following should suggest the condition to the clinician; an anemic patient who gives a history of vague abdominal distress and who further states that on several occasions he has vomited material looking much like egg white. He may or may not give the history of hematemesis and diarrhea. The finding of an achlorhydria upon the examination of the gastric secretions, along with excessive mucus, further substantiates the diagnosis. A mottled, fingerprint, filling defect in the lumen of the stomach seen on the roentgenogram and the direct visualization of multiple polyps on the gastric mucosa with the gastroscope is a positive confirmation of the diagnosis.

TREATMENT

Gastric polyposis has been treated in many ways with greater and lesser degrees of success. Douglas treated one patient by dropping a capsule of radium, tied to a string, into the stomach. This was left in the stomach for six hours with frequent changes in the position of the patient. Patient died three months later. Therapeutic roentgen ray treatment has been used with the relief of symptoms for variable periods of time. However, the symptoms return and are commonly increased in severity. It is impossible to tell whether the temporary relief of symptoms was due to the therapy or whether it was merely a spontaneous remission which frequently occurs. Cases of natural cure have been reported, in which torsion or strangulation of the pedicles have separated the polyps from their bases. It is generally accepted at present that the treatment of choice is surgery. Three procedures have been used; (1) local excision of the polyps through a gastrostomy; (2) gastric resection; and (3) gastric resection with the excision of a few remaining polyps in the gastric stump. Brunn and Pearl excised the polyps locally in four benign cases, achieving good results in only two of the four cases. They also carried out the same procedure in four malignant cases. Of these four, two patients were relieved for a period of five years, after which time there was a recurrence of symptoms. It can easily be seen, therefore, that local excision is grossly inadequate. The same men did gastric resections on five benign cases, and satisfactory results were obtained in all but one. The superiority of gastric resection over local excision is readily appreciated. Probably more important is the fact that gastric resection eliminates the possibility of malignant degeneration.

PROGNOSIS

Many cases of gastric polyposis remain benign and asymptomatic for years. However, all cases diagnosed should be treated, as soon as the patient is satisfactorily prepared, by gastric resection. If malignancy has not occurred, the prognosis is excellent following gastric resection. If malignancy has occurred, gastric resection is the most effective method of treatment. That all gastric polypi are potentially malignant should be constantly kept in mind.

SUMMARY

A brief history of gastric polyposis has been outlined. The etiology, pathology, malignant changes, signs and symptoms, roentgen and gastroscopic examination, diagnosis, treatment, and prognosis have been discussed in detail.

CONCLUSIONS

Gastric polyposis is a rare condition occurring most frequently during the fourth and fifth decades in men more often than in women. There are two accepted theories of origin; the congenital and the inflammatory or acquired, the congenital theory being the more valid of the two. Two types of polyps occur; the polyadenome polypeaux, and the polyadenome en nappe, the former being hypertrophic in character, and the latter hyperplastic. The signs and symptoms are in no way pathognomonic of the condition, and the diagnosis should only be made after proper roentgen and gastroscopic examination. Since all cases are potentially malignant the treatment of choice is gastric resection. The prognosis is excellent in cases treated in this manner.

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WELL DESERVED HONORS

Elsewhere in the Journal is printed a list of the physicians of the state who have received from General Fleming of Selective Service a citation and a medal for the work on the draft boards that they did as civilians during the war. The medals and citations are well deserved rewards for the time and effort that were given voluntarily by a group of physicians to help the war effort. Many of these men for the entire duration of the war gave an untold number of hours to the examination of the man who was a candidate for induction into the army. At the beginning of the war a tremendous amount of time was contributed by most of these men to the examination of the young man who had been drafted. Hours and hours of purely voluntary effort were devoted to the tiresome and laborious routine examination of innumerable men. Later in the war the demands were not quite as strenuous but even then many doctors cheerfully and willingly gave of their time, extracted from the inordinate demands of wartime with a markedly reduced physician complement. The men to whom these medals have been awarded can feel proud to pass them on to their children and grandchildren to show that while they were in civil life they nevertheless helped make our war efforts successful.

It will be noted that many of the men to whom the medals have been awarded subsequently entered the armed services, and further gave of their services full time to their Country. It will be noted also that quite a few of the recipients of the medal have died. This illustrates well indeed the fact that it was the older man who was called upon for this particular service. We feel sure that having to take over the practice of other doctors, having to give time to this type of routine work, and having little time to care for themselves, was responsible for the death of some of the physicians who passed away during the war.

Incidentally the medals can be preserved and hung on the wall of the office by having them framed appropriately. The medals could well grace the office of doctors who so cheerfully, willingly and unselfishly gave their time to the type of work which at best could not be called exhilarating or stimulating.

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WAGNER-MURRAY-DINGELL BILLS

Numerous editorials have appeared in the Journal under the above caption and probably there will be more. It does seem more or less ridiculous to keep harping on one subject but when all is said and done this bill, or bills, are of fundamental and basic importance to the doctor. If the medical profession for one minute relaxes their effort to defeat this particular legislation it is quite possible that it might be slipped over without their being aware of just what is going on at the moment. This, however, is doubtful but there is no doubt that we must be eternally vigilant.

At the present there is not very much likelihood of any immediate action being taken on these bills as Senator Murray has announced that until Congress reconvenes in the fall hearings on the bills will cease.

Comment has been made previously on the reporting of the hearings that are appearing in the Journal of the American Medical Association. If the busy practitioner has time to read the abstracted hearings presented before the Senate Committee on Education and Labor he will be struck by several features that well deserve comment. In the first place the opposition to the bills, led largely by the medical profession, seems to have so many arguments against the proposed legislation that are valid, accurate and well substantiated that it would seem as if the medical opponents of the bills have all the best of the argument. Of course the disinterested observer might say that this is the opinion of a physician but we feel sure that were the report of the hearings read by disinterested observers as for example, an English physician or a Norwegian doctor, or a Mexican barrister, or an Australian lawyer, they would agree with the statement that has just been made.

The proponents of the bills, on the other hand, seem to be very largely moved by sentiment. It is a very easy thing to appeal to sentiment, more particularly when it comes to such sentimental subjects as sick children. It is all right for such a charming and able physician as Dr. Martha Elliot to say that "either we spend money or we spend children." Does Dr. Elliot believe for a minute that the spending of very large sums of money is going to improve materially a situation in which maternal deaths, for example, are numbered only 24 to 10,000 mothers and the infant mortality of 40 deaths per 1,000 live births? Dr. Elliot has testified to the effect that every day the passage of the Wagner-Murray-Dingell bills is delayed is responsible for the death of large numbers of children. Again, we doubt very much if such a statement could be substantiated. We do not believe that many of these children would be saved even if they were treated by the professor of obstetrics at Johns Hopkins or Harvard University or by the professor of pediatrics at Columbia or Northwestern University. Statements such as Dr. Elliot has made are not convincing to the thoughtful individual although they do appeal to those who are hypersensitive and oversentimental.

Senator Murray, in his published comments on the bill that bears his name, states that one of the pressing reasons for this particular bill is because the indigent are dying without proper medical care. That is another statement that could be very well refuted. The indigent in large cities receive care which is comparable to that which may be given to a millionaire. Certainly in the rural sections, at least in Louisiana, the indigent are being treated at our big state hospitals by outstanding members of the medical profession. They receive medical care and attention which is notable. With good roads and the easy accessibility to even the most remote country districts it is possible to get a patient, for example, in a charity hospital within a very few hours. Furthermore the smaller state hospitals are wont to refer their difficult diagnostic and therapeutic cases to the two big state hospitals which, in equipment, lack nothing and whose medical personnel is highly skilled. If these revolutionary bills should be passed it is certainly not for the benefit of the extremely poor.

It is difficult indeed for any physician or for an intelligent layman to understand why medical services which are acknowledged to be the best in the world should suffer by the tremendous upheaval and disturbance in medical practice which the passage of the Wagner-Murray-Dingell bill is bound to occasion.

CORONARY ATHEROSCLEROSIS

Coronary disease is responsible for the death of more than one in five doctors in this country. Twice as many doctors die of coronary disease as die of cancer. Probably if studies were made or statistics kept in regard to the clergy, the law profession or to teachers the same general ratio would probably exist. The principal cause of death in men in this country at the period of their highest earing capacity is due to the pathologic involvement of a few grams of coronary intima. William Dock has made these statements in a recent paper in the Journal of the American Medical Association*. Dock speaks with authority. He is now professor of pathology at Cornell University Medical School and in the last several years has been professor of medicine at two prominent medical schools and professor of pathology at two other institutions. Many Louisiana physicians should have a vicarious interest in this author. His father some thirty-five years ago was professor of medicine at Tulane and is one who, despite the passage of eighty some years, still leads an active and vigorous existence.

The younger Dock has been interested for many years in coronary disease. He has recently called attention to the large number of young soldiers who die as a result of coronary atheroma while training in the United States. Many of these young men showed no evidence of arterial disease elsewhere. There are two predisposing factors of great importance in the etiology of atherosclerosis, to wit, hypertension and hypercholesteremia. In the younger man the latter factor is the more important of the two. The soldiers of the United States were the best fed soldiers in the world and their diet included butter, ice cream, milk, eggs; all substances high in cholesterol which might have played a factor in producing the cholesterol changes which were found in the coronary arteries of these young men. In people whose diet is cholesterol poor, but rich in whole grain, legumes and vegetable oils, coronary disease seems to be a rarity.

Probably one of the most remarkable features of coronary infarction or occlusion is the frequency with which it attacks men. It is only the occasional woman that suffers from this condition whereas, as noted before, it is extremely common in men. One of the important features of Dock's paper lies in the fact that he offers an explanation for the preponderance of males dying of coronary disease. This explanation is no hypothesis but is a definitely proved observation. Dock shows that, in the first place, the predilection of atheroslerosis of the coronary arteries is because they possess an intima which varies in thickness from ten to six hundred per cent of that of the media in the newborn infant. Secondly in the infant male whose intima was accurately measured, the average is 26 per cent whereas in the female infant it is only eight per cent.

Dock's paper is short, succinct and informative. It offers an explanation, in a very few pages, for two of the important problems of coronary disease which have not been previously answered: Why is the coronary artery more frequently involved than other relatively small arteries throughout the body and why atherosclerosis is so much more common in the male than in the other sex. Parenthetically it might be stated that Dock believes that the present appalling death rate in coronary disease may in the future become reduced as our understanding of hypertension and cholesterol metabolism becomes better understood. He even suggests the possibility that the incidence of coronary disease may fall to the insignificent level which is observed in the Chinese.

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*Dock, William: Coronary atherosclerosis, J. A. M. A., 131:875, 1946.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 MEETING

Word has been received from the New Orleans Hotel Association that ample accommodations will be available for registrants at our meeting next year on May 12-14. Headquarters have not yet been selected for the meeting, however Dr. Edwin L. Zander has been appointed chairman of the Committee on Arrangements and important details will be handled promptly and ably under his guidance.

The Committee on Scientific Work, the special Committee to Consider Rearrangement of the Program and the chairmen of the various scientific sections will meet within the next few weeks to work out plans for a scientific program which will prove of interest and benefit to members attending the meeting.

STATE LEGISLATION

During the past session of the Louisiana Legislature two of the most important bills which the medical profession sponsored, after being passed by the senate and the house, were vetoed by the governor. Both of these bills called for additional expenditures which unquestionably influenced the veto owing to the fact that the legislature had appropriated money far beyond the state income. After fighting for several years for a state mental hospital close to New Orleans to take care of the tremendous number of patients at the other state institutions and after being successful in having the measure approved by both houses the appropriation of $300,000 was vetoed by the governor because a two-thirds majority did not prevail at the time action was taken.

Similar action befell the attempt to provide for needed statewide facilities for the chronically ill adjacent to our state hospitals. This bill was supposed to have had the endorsement of the administration and other powerful groups in addition to the medical profession, however after passing both houses it was finally vetoed by the governor for the same reason as for the mental hospital bill. Obviously owing to the exigencies of war there is an increased cost of maintenance, food, medical and special supplies for our present institutions which required increased appropriation for the already existing institutions. This increase had not been anticipated sufficiently to warrant the added expenditure of money far beyond the state income.

NATIONAL LEGISLATION

From a national viewpoint it will be interesting to note that from all information that we have received from accredited and reliable sources, Senate Bill 1606, known as the National Health Program, and featuring compulsory health insurance, will not be considered by the 79th Congress. You should know that it is regarded as a sound opinion that the satisfactory results obtained in this regard were due to the coordination and support of the medical profession of this country, and especially to the activities of our various state medical societies under the guidance of astute leaders in Washington and Chicago. These men represented the American Medical Association through the Council on Medical Service and Public Relations and through the National Physicians Committee. We certainly wish to commend them for the wonderful job which they have performed, not merely for the medical profession but for the public at large.

Apparently some members of the Committee on Education and Labor which has S. 1606 for consideration, felt that they would give some substantial support to this measure by stating in the press that they are in favor of some compulsory medical insurance, not controlled by the government. However this is rather misleading and one will have to be influenced entirely by future action of this committee.

Please do not misunderstand the above statements as in anyway indicating that the question of federalization of medicine is a dead issue. Certainly in some form or fashion even if not considered by the 79th Congress it will be brought up in the 80th Congress after January 1, 1947.

There are certain things which have developed incident to these hearings before the Committee on Education and Labor. There was an attempt made by Senator Pepper on July 5 and July 15 to bring up before the Senate S. 1318, known as the Pepper Maternal and Infant Care bill. He failed in this attempt but undaunted he, along with Senator Taft who is also a member of the Committee on Education and Labor (and a reliable opponent to S. 1606) introduced in the Senate on July 15 a joint resolution No. 177 which was referred to the Committee on Finance. This joint resolution is an amendment to Title V of the Social Security Act of 1935, whereby $31,500,000 is appropriated instead of $5,873,000 appropriated in the original act for maternal and child health services. If this joint resolution prevails and an increased appropriation is granted it would, according to reliable critics, simply mean that we would go back to the year 1935 and consider all maternal and infant care services on a state basis, under the supervision and control of the state boards of health.

Senators who are members of the Committee on Education and Labor have sent us telegrams advising that action on S. 1318 has been postponed.

According to the action taken by our House of Delegates in May a communication was sent to every member of the State Society on May 30 advising that the agreement between our organization and the State Board of Health in regard to the EMIC plan, had been terminated, and asking that members of the State Society refuse to take further cases under this apprehension. Subsequent to this action the Executive Committee approved acceptance of cases where the patients were pregnant prior to May 8. Notice of this was carried in the organization section of the July issue of the Journal.

However, careful as we have tried to be in notifying our members of the above action, we are informed by the State Board of Health that some physicians are still making application to the Board of Health for remuneration for maternal and infant care under the old plan in existence during the war. Every member of the State Society is respectfully admonished not to accept any further cases under the EMIC program except as provided for by the Executive Committee, in order that the Board of Health may not be misled as to the desires of organized medicine in this state.

Recently, President Truman proposed extensive reorganization steps for the Executive Department of the federal government. Contained in his proposal was the removal of the Children's Bureau from the Department of Labor to the Federal Security Agency. Under existing statutes, the failure of either House of Congress to oppose this phase of the President's reorganization plan, the Children's Bureau was permitted to move into the Federal Security Agency.

It will be of interest to members of our organization to know that efforts are being made to have a uniform contract with the Veterans Administration throughout the state. This will enable veterans to be treated in their home towns by their own doctors and should prove to be a very satisfactory arrangement for the veteran and the physician.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Aug. 14. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.

Aug. 15. Clinico-pathologic Conference, Touro Infirmary, 12 noon.

Aug. 22. DePaul Sanitarium Staff, 8 p. m.

Aug. 23. L. S. U. Faculty Club, 8 p. m.
Ochsner Clinic Staff, 8:30 p. m.
Aug. 26. Board of Directors, Orleans Parish Medical Society 8 p. m.
Aug. 28. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Aug. 29. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Aug. 30. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Sept. 3. Orleans Parish Radiological Society, 7:30 p. m.
Sept. 5. Executive Committee, Baptist Hospital, 8 p. m.

EVERY TUESDAY, Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

EVERY WEDNESDAY, Medical Discussions, Radio Station WDSU (1280 on your dial).

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NEWS ITEMS

At the July meeting of the Society a motion picture entitled Scalp Avulsion was shown. At this meeting Dr. C. J. Vedrenne presented a paper on the Pathogenesis of Postoperative Atelectasis.

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Dr. Donovan C. Browne attended the recent meetings of the American Gastro Enterological Association, and the American Gastroscopic Club at Atlantic City.

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Dr. William B. Clark recently presented a preliminary report of his work on onchocerciasis in Guatemala before the Section of Opthalmology of the New York Academy of Medicine.

While in New York Dr. Clark took a two weeks' course in the surgery of keratoplasty under Dr. D. Townley Paton, director of the National Eye Bank, at the Manhattan Eye and Ear Infirmary, with the object in view of establishing a branch of the National Eye Bank in New Orleans.

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Drs. Anna D. Colomb, Hunter P. Harris, Walter J. Otis and Walker Thompson attended the recent meeting of the American Psychiatric Association at Chicago.

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Dr. Henry O. Colomb recently attended the meeting of the American College of Physicians at Philadelphia.

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Dr. E. Carroll Faust was presented with a "Medal of Freedom". This award was given by the war department in recognition of his work on schistosomiasis in the Philippines.

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Dr. Daniel J. Murphy recently addressed the Louisiana Pharmaceutical Association. The subject of his address was, "Medicine's Tribute to Pharmacy."

Dr. Murphy was appointed by Mayor deLesseps S. Morrison as a member of the Advisory Committee for Veterans' Affairs.

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Dr. William A. Sodeman gave a series of six talks on internal medicine during the annual graduate short course for doctors in Jacksonville, June 17-22. The course was under the joint sponsorship of the University of Florida and the Florida state board of health.

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Dr. Julius Lane Wilson attended the meeting of the National Tuberculosis Association at Buffalo, June 10-13.

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The regular monthly meeting of the Journal Club of L. S. U.'s Department of Obstetrics and Gynecology was held June 5 at the Jung Hotel. Dinner was served first, after which several interesting papers on diabetes in pregnancy were given by members of the L. S. U. obstetric and gynecology resident staff of Charity Hospital. Members of the L. S. U. internal medical staff also contributed to the discussion. The meeting was attended by the visiting physicians, residents and interns of the L. S. U. obstetrics and gynecology unit of Charity Hospital. Guests included physicians from other services of the hospital and school.

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NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

At a recent meeting of the New Orleans Graduate Medical Assembly the following physicians were elected to office:

Dr. William H. Gillentine, president; Dr. Joseph S. D'Antoni, president-elect; Dr. Randolph Lyons, first vice-president; Dr. H. Ashton Thomas, second vice-president; Dr. C. S. Holbrook, third vice-president; Dr. Max M. Green, secretary; Dr. Edwin H. Lawson, treasurer; Dr. C. Barrett Kennedy, director of program; Drs. J. O. Weilbaecher, Jr., and Ambrose H. Storck, assistant directors of program; Drs. L. C. Chamberlain, Frederick F. Boyce, Lucien A. Fortier, Howard R. Mahorner and Charles B. Odom to the executive committee.

The tenth annual meeting of the Assembly will be held February 24-27 with headquarters at the Municipal Auditorium.

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WELCOME HOME

The following physicians were recently separated from military service: Dr. John J. Archinard, Ochsner Clinic; Dr. John G. Atwood; Dr. George G. Gill, Ochsner Clinic; Dr. John G. Menville, 606 Maison Blanche Bldg.; Dr. Carl N.

Wahl, 825 Maison Blanche Bldg.; Dr. Daniel W. Beacham, Charity Hospital; Dr. Michael N. Goldberg, Charity Hospital; Dr. Frederick L. Reuter, Charity Hospital; and Dr. John O. Redding, Jr., removed to Asheboro, N. C.

CALLED TO SERVICE

On June 10 Dr. J. William Rosenthal entered the medical corps of the United States Naval Reserve.

CORRECTION

In the last issue of the Journal we inadvertently listed Dr. E. L. Leckert, Jr., as having been called to military service as a *second* lieutenant. Dr. Leckert is a *first* lieutenant in the United States Army. We offer our apologies.

CHANGES OF ADDRESS

Dr. Irvin Cahen, 1441 Delachaise Street; Dr. John T. Crebbin, 1857 Benefit Street; Dr. G. Nelson Furbeck, Gante 1, Mexico, D. F. Mexico; Dr. John E. Garcia, 1305 Canal Building; Dr. W. Robyn Hardy, 601 Maison Blanche Building; Dr. Henry J. Lindner, Veterans Hospital, Lyons, N. J.; Malcolm F. Schwarzenbach, 1631 Murl Street; Lt. Jack R. Anderson, U. S. S. Howard Gilmore (AS-16), Fleet Post Office, New York; Lt. Thos. E. Furlow, U. S. Navy Recruiting Station, Dallas, Texas.

CHANGES IN MEMBERSHIP ROLLS

Four intern members were transferred to active membership—Drs. Wm. Fisher, Louis Levy, II, Robert M. Rose and Harry Rollings.

Two associate members were transferred to intern membership—Drs. Michael N. Goldberg and Frederick L. Reuter.

Transferred from active to inactive membership—Dr. James T. Nix.

Removed—Active members: Drs. Arthur J. Butt, Jr., Julius R. Fernandez, E. Borland Gill, John G. Pratt, Richard Selser, Miriam O. Vinnard, and Edgar Warren; associate members: Drs. Louis L. Friedman and John G. Hamilton; intern members: Drs. Howard M. Bordelon, Robert O. Harris, III, Frank S. Oser, Jr., O. L. Pollingue, Jr., John C. Taylor, Jr., Kendrick Taylor, Levon F. Wright and James H. Young.

RADIO PROGRAM

The following "Health Discussions" will be broadcast over radio station WDSU (1280 on your dial) during the month:

July 10th—Hypertension—Dr. Thomas Findley.

July 17th—Rabies in New Orleans—Dr. George H. Hauser.

July 24th—Polio—A Vacation Threat—Dr. Guy A. Caldwell.

July 31st—Infectious Diseases in Children—Dr. Frank Kinberger.

August 7th—Acute and Chronic Appendicitis—Dr. Daniel J. Murphy.

WELCOME HOME PARTY

On Monday, June 24 the Society officially welcomed home its members who served their country in World War II. A supper which was well attended, was held in the Ball Room of the New Orleans Athletic Club.

Dr. Daniel J. Murphy, president of the Society, delivered the following address of welcome: "Tonight our Society takes this opportunity to officially welcome home those of its members who so nobly answered the call of our country in the second World War. It is happy and proud to have upon its rolls so many names that have covered themselves with glory. As the president, it gives me so much satisfaction, in the name of the Orleans Parish Medical Society, to say "*Welcome Back*". To me, no lengthy speech could possibly convey to you our pleasure in having all of you returned safe and sound to us. We are fortunate in the fact that our Society did not lose a single member as a war casualty. For this we are happy and are thankful. Fortunate as we are in not losing a member, some of the lustre of our happiness is dimmed when we think of the members of our Society who suffered the loss of some of their dear ones. To them our sympathy is extended and we hope that they can draw some measure of consolation in the thought that our nation is forever grateful to their departed ones for having made the supreme sacrifice so that all of us may live in peace. No flowery speech, no display of dramatics, no eloquence in oratory can possibly express to you our joy in having all of you returned to us. So in the name of the Society, I know of no better way to show our appreciation to you for what you have done for us and the pleasure we have in your safe return than by saying "*Welcome back and thank God that you are back again with us to stay!*"

Dr. Edmond Faust responded for the Army; Dr. Shirley C. Lyons for the Navy; Dr. John Oakley for the Marines; Dr. Samuel Colvin for the Tulane Hospital Unit; Dr. Charles Miangolarra for the L. S. U. Hospital Unit.

Between each response the theme song of that branch of service was sung; accompaniment at the piano was by Dr. Emile A. Bertucci.

The meeting closed with the singing of the Star Spangled Banner.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SELECTIVE SERVICE AWARDS

Doctors serving on Medical Advisory Boards in Louisiana who have received the Selective Service Medal:

Drs. Maurice Campagna, Ernest C. Samuel, Julius Wilson, G. C. Anderson, H. F. Brewster, Edmund Connely, Val H. Fuchs, Edgar Hull, S. C. Jamison, D. Urban Maes, Leon J. Menville, J. R. Schenken, H. T. Simon, B. J. DeLaureal, W. H. Harris, C. S. Holbrook, F. E. LeJeune, Randolph Lyons, E. Alton Oschner, J. T. O'Ferrall, Victor C. Smith, T. A. Watters, E. B. Vickery, Charles A. Bahn, L. A. Fortier, Lewis Golden (Deseased), Roy B. Harrison, Sam Hobson, E. H. Lawson, J. H. Musser, H. W. E. Walther (Deseased), all of New Orleans.

Drs. Dean H. Duncan, H. A. Durham, L. W. Gorton, W. R. Harwell, Joe E. Heard, Douglas L. Kerlin, W. Sam Kerlin, Morgan W. Matthews, W. R. Mathews, I. B. Rougon, John L. Scales, all of Shreveport.

Drs. A. J. Comeaux, Lafayette; L. O. Clark, Lafayette; C. C. DeGravelles, New Iberia; C. E. Hamilton, Lafayette; Paul H. Kurzweg, Jr., Lafayette; L. B. Long, Lafayette; Jos. D. Martin (Deceased), New Iberia; John M. Miles, Lafayette; J. D. Frazar, DeRidder.

Drs. S. F. Hatchette, Louis A. Hebert, R. G. Holcombe, D. C. Iles, G. C. McKinney, O. W. Moss, and T. H. Watkins, all of Lake Charles; J. Q. Graves, C. P. Gray, W. M. Hunter, C. P. Jarrell, D. M. Moore, P. L. Perot, and John Pracher, all of Monroe; H. O. Barker, M. H. Foster, M. S. Freiman, O. B. Owens, P. K. Rand, N. T. Simmonds, and B. M. Wilson, all of Alexandria; William B. Chamberlin, U. S. Hargrove, Brinsfield King, L. F. Lorio, T. Jeff McHugh, T. H. Pargen, Lester J. Williams, and Howard B. Witter, all of Baton Rouge.

To the following were also awarded medals and certificates:

Ascension Parish: Drs. Forest E. Baker, Gonzales; Henry A. Folse, Donaldsonville; Gerald L. Gaudin, Gonzales; Earl A. Schexnayder, Donaldsonville.

East Baton Rouge Parish: Drs. W. L. Comeaux, Oswald W. Cosby, W. A. Dial, W. R. Eidson, Frank W. Pickell, Felix Boizelle, J. P. Griffon, J. R. Godfrey, H. C. Hatcher, W. K. Irwin, Rhett G. McMahon, J. D. Martin, Jr., H. Guy Riche, Ashton Robins, Charles H. Voss, D. D. Barnett, W. H. Cook, F. U. Darby, L. J. Kern, Jr., H. W. A. Lee, Jos. J. Noto, Philip B. Polito, William McHugh Simmons, W. B. Singletary, W. Shewen Slaughter, J. A. Tucker (Deceased), B. E. Nelkin, and C. O. Bingham, all of Baton Rouge.

East Feliciana Parish: Dr. C. S. Toler, Clinton.

Iberville Parish:Drs. William E. Barker, Jr., J. Cyril Eby, Adrian Landry, and Eugene Holloway, all of Plaquemine.

Rapides Parish: Drs. R. O. C. Green, S. L. Calhoun, J. T. Cappel, A. L. Culpepper, J. P. Durham, H. P. Forsyth, H. H. Hardy, Ralph Lampert, L. F. Magruder, R. G. Masterson, R. E. C. Miller, W. M. McBride, R. U. Parrott, G. W. Smith, R. B. Wallace, Harry Koretsky, James W. Welch, M. B. Pearce, N. M. Brian, Jr., V. F. Chicola, J. H. Landrum, R. E. McGill, W. L. Murrell, and E. R. Pincus, all of Alexandria. Dr. B. F. Bremer, Glenmore. Dr. R. C. Scott, Lecompte.

Caldwell Parish: Drs. E. L. Carroll and D. O. Sherman, Columbia. Dr. J. G. Norris, Farmerville.

East Carroll Parish: Drs. D. F. Davis, and J. Preston Davis, Lake Providence.

Franklin Parish: Drs. L. F. Robinson, and J. D. Rogers, Winnsboro.

Jackson Parish: Drs. T. A. Dekle, M. M. Hearne, and C. S. McDonald, Jonesboro. Dr. George H. Robinson, Hodge.

Catahoula Parish: Dr. P. W. Calliham, Harrisonburg.

Concordia Parish: Drs. A. J. Hodges, and Joseph H. Pankey, Ferriday.

Grant Parish: Dr. Nathaniel M. Brian, Montgomery. Dr. D. V. Donaldson, and J. F. Woodall (Deceased) Colfax. Dr. J. W. Scott, Rochelle.

Lasalle Parish: Dr. E. L. Miller, Jena. Dr. F. A. Thomas, Urania. Dr. David Trax, Trout.

Natchitoches Parish: Drs. R. E. Corkern, W. M. Knipmeyer, M. H. Phelps, W. H. Pierson, R. S. Roy, all of Natchitoches.

Vernon Parish: Dr. E. E. Archibald, Alco. Dr. M. S. Stephens, Hornbeck. Drs. W. E. Reid, E. M. Shaw, and M. W. Talbot, Leesville.

Winn Parish: Drs. J. F. Faith, S. C. Fittz (Deceased), and J. T. Mosley, all of Winnfield.

Livingston Parish: Drs. A. A. Murray, and Montgomery Williams, Denham Springs.

Pointe Coupee Parish: Drs. E. G. Durel, J. M. Mosely, J. W. Plauche, Jr., and F. F. Rougon, all of New Roads.

St. Helena Parish: Dr. William E. Day, Greensburg.

Tangipahoa Parish: Drs. A. L. Lewis, and M. B. Small, Amite. Dr. Luther L. Ricks, Independence. Drs. R. G. Korniker, and G. T. Scott, Jr., Ponchatoula. Drs. Alexander J. Feder, C. D. Overton, M. C. Wigington, Carlos D. Alessi, Collins P. Lipscomb, and John D. Thomas, all of Hammond.

West Baton Rouge Parish: Drs. D. W. Landess, and Paul B. Landry, Port Allen.

West Feliciana Parish: Drs. Cecil C. Blakeney, and Philip A. Niebergall, St. Francisville.

Avoyelles Parish: Drs. A. B. Cairns, and H. C. Jones, Bunkie. Dr. J. C. Goldner, Marksville. Dr. Philip Jeansonne, Plaucheville. Dr. Kirby A. Roy, Mansura.

Lincoln Parish: Drs. Marvin T. Green, J. B. Harris, S. L. White, and E. J. Young, Ruston. Dr. Jos. L. Smith, Dubach.

Madison Parish: Drs. D. H. Allen, F. A. DeJean, E. Otis Edgerton, G. W. Gaines (Deceased), Howard Clay Sevier (Deceased), and William L. Yerger (Deceased), all of Tallulah.

Morehouse Parish: Drs. William V. Garnier, John Newton Jones, Rae Byrd Leavell, W. A. Rodgers, and Smith I. Sims, all of Bastrop. Dr. E. L. Maddry, Oak Ridge. Guy D. Williams, Mer Rouge.

Ouachita Parish: Drs. H. V. Collins, Henson Coon, A. E. Fisher, J. T. French, Henry E. Guerriero, C. H. Hill, C. U. Johnson, W. E. Jones, John G. Snelling, Jr., George Varino, Irving J. Wolff, Glenn T. Gallaspy, and Ellis Powell, all of Monroe.

Richland Parish: Drs. John C. Calhoun, and H. C. Chambers, Rayville, Drs. Sheldon Teer, and C. C. Thompson, Delhi.

Tensas Parish: Dr. Joseph Whitaker, St. Joseph.

Union Parish: Dr. C. C. Colvin, Bernice. Drs. C. C. Francis, and W. P. Lambeth, Farmerville.

West Carroll Parish: Dr. Daniel W. Kelley, Oak Grove.

Allen Parish: Drs. Gurdon Buck, Kinder. Dr. E. H. Kent, Oberlin. Dr. A. D. Mangham, Clarks. Drs. L. F. Gray, M. V. Hargrove, and W. Rigsby Hargrove, all of Oakdale.

Beauregard Parish: Drs. Luke Marcello, S. T. Roberts, and Thomas R. Sartor, all of DeRidder.

Calcasieu Parish: Drs. B. D. Atchley, T. B. DeLaurel, Willie G. Fisher, Ben Goldsmith, Fritz LaCour, Walter O. Moss, L. L. DiGiglia, R. P. Kandle, J. G. E. Barham, all of Lake Charles. Dr. A. H. Forman, Vinton. Dr. George S. Lyons, DeQuincy. Drs. Robert B. Marshall, and W. A. K. Seale, Sulphur.

Cameron Parish: Dr. J. B. Colligan, Hackberry.

Jefferson Davis Parish: Drs. Trois E. Johnson, Richard S. Kramer, Louis E. Shirley, Morgan Smith, F. W. Harrell, all of Jennings. Drs. Claude Martin, and John G. McClure, both of Welsh.

Acadia Parish: Dr. C. D. Brunt, Iota. Drs. J. W. Faulk, H. L. Gardiner, S. R. Henry, A. R. Morgan, E. S. Peterman, all of Crowley. Drs. John S. Baily, and T. T. Tolson, both of Church Point. Drs. Ernest C. Faulk, and R. C. Webb (Deceased), both of Rayne.

Evangeline Parish: Dr. R. E. Dupre, Ville Platte. Dr. O. L. Freeman, Basile. Dr. Frank Savoy, Mamou. Dr. Arthur Vidrine, Ville Platte, and Dr. G. G. Fontenot (Deceased), Ville Platte.

Iberia Parish: Drs. L. A. Breffeilh, Lewis B. Crawford, Orien E. Dalton, Henry J. Dauterive, Harold M. Flory, Edwin L. Landry, and Wilton P. Tilly, all of New Iberia. Dr. Wilfred Morgan, Weeks. Drs. C. W. Phillips, and Lastie M. Villien, both of Jeanerette.

Lafayette Parish: Drs. J. O. Duhon, Clarence J. Saloom, and C. K. Olivier, all of Lafayette.

St. Landry Parish: Drs. Lionel J. Bienvenu, W. R. Lastrapes, S. J. Rozas, Oscar J. Bienvenu, Ertemon Lafleur, all of Opelousas. Dr. George A. Azar, Washington. Dr. Leo L. Collins, Port Barre. Drs. Harry Jenkins, Charles W. Lewis, and Weston R. Miller (Deceased), all of Eunice. Dr. Gordon Morgan, Melville. Dr. Dewitt Grover, Rackley.

St. Martin Parish: Drs. J. L. Beyt, A. R. Corne, and P. H. Fleming (Deceased), all of St. Martinville. Drs. L. A. Morrogh, and S. D. Yongue, both of Breaux Bridge.

St. Mary Parish:Dr. J. C. Berwick, Berwick. Drs. T. H. Gueymard, and F. H. Metz, both of Morgan City. Dr. J. R. Rushing, Baldwin. Drs. A. C. Kappel (Deceased), L. J. Supple, and G. Vasquez, all of Franklin.

Vermilion Parish: Dr. L. M. Boudreaux, Erath. Dr. George L. Gardiner, Sr., Gueydan. Drs. Thomas Latiolais, Edward R. Villemez, and Leo Saporito (Deceased), all of Kaplan. Drs. J. E. McClellan, Shelley Mouledous, Marion A. Young, Jr., and L. E. Chauvin, all of Abbeville.

Bienville Parish:Dr. V. W. Fletcher, Ringgold. Drs. Orum L. Kidd (Deceased), and Carl L. Langford, both of Gibsland. Dr. J. L. Wade, Arcadia.

Bossier Parish: Drs. H. N. Barnett, and J. B. Hall, Jr., both of Benton. Dr. D. C. McCuller, Bossier City.

Caddo Parish: Drs. W. B. Allums, Dorf Bean, Thomas V. Bush, F. A. Buvens, L. H. Pirkle, E. L. Sanderson (Deceased), Thomas R. Simpson, R. R. Wilson, Wm. M. Hall, G. H. Cassity, I. F. Hawkins, C. H. Potts, H. J. Quinn, W. J. Sandidge, Ernest B. Flake, W. B. Heidorn, Clifford R. Mays, T. J.

Smith, J. Dudley Talbot, J. M. Bodenheimer, and R. B. Delee, all of Shreveport. Dr. Matthias M. Collins (Deceased), Hosston. Drs. J. W. Mobley, and C. H. Pardue, both of Vivian. Dr. William S. Siler, Jr., Ida. Dr. T. B. Tooke, Belcher.

Claiborne Parish: Drs. E. A. Campbell, and B. L. Stinson, both of Homer. Drs. A. G. Worley, and W. L. Waller, both of Haynesville.

Desoto Parish: Drs. W. B. Hewitt, and R. A. Tharp, both of Mansfield.

Red River Parish: Dr. Jack Carlisle, East Point. Dr. L. S. Huckabay, Harmon. Dr. W. B. Hunter, Coushatta.

Sabine Parish: Drs. S. F. Fraser, J. K. Phares, and Gustave F. Weber, all of Many. Dr. R. L. Parrott, Zwolle. Dr. H. M. Prothro, Pleasant Hill.

Webster Parish: Drs. Claude M. Baker, J. B. Benton, S. F. Martin, and R. E. Smith, all of Minden. Dr. W. C. Gray, Springhill. Dr. John Pugh, Cotton Valley.

Assumption Parish : Dr. Dewey Billeaudeaux, Klotzville. Dr. Julius W. Daigle, Belle Rose. Dr. Henry A. LeBlanc, Paincourtville. Drs. Nelson A. Cox, W. E. Kittredge (Deceased), William Pugh, and C. S. Roger, all of Napoleonville.

Jefferson Parish: Drs. John W. Atkinson, Carroll F. Gelbke, Charles Gelbke, and Floyd M. Hindelang, all of Gretna. Dr. Philip P. LaBruyere, Marrero. Dr. Joseph J. Massony, Westwego. Dr. L. J. Genella (Deceased), Kenner. Dr. J. S. George, Metairie.

Lafourche Parish: Drs. Charles J. Barker, F. T. Beatrous, J. L. Danos, and Murphy P. Martin, all of Thibodaux.

Plaquemines Parish: Dr. Benj. H. Carlton, Port Sulphur. Dr. B. R. Slater, Belle Chasse.

St. Bernard Parish: Drs. A. V. Filizola, Felix Planche, N. P. Trist, Jr., all of Arabi.

St. Charles Parish: Drs. Samuel L. Gill, and Roy A. Kelly, both of Destrehan. P. T. Landry, Norco.

St. James Parish: Dr. Stephen R. Campbell, Hymel. Dr. Anthony J. Nobile, Lutcher. Dr. Harry J. Schmidt, Convent. Dr. L. O. Waguespack, Vacherie.

St. John Parish: Drs. Pierre A. Donaldson, and J. S. Parker, both of Reserve. Dr. J. R. Fernandez, Wallace. Drs. Remy F. Gross, and W. F. Guillotte, Laplace

St. Tammany Parish: Drs. H. E. Cannon, and H. E. Gautreaux, both of Covington. Dr. F. R. Singleton, Slidell.

Terrebonne Parish: Drs. S. Clark Collins, J. B. Duval, S. Ernest Ellender, W. A. Ellender, H. L. Haydel, M. F. Houston, and H. P. St. Martin, all of Houma.

Washington Parish: Drs. J. S. Desporte, W. A. Fleming, W. S. Harrell, E. E. Lafferty, Frank Mock, and A. G. Murphy, all of Bogalusa. Drs. W. R. McGehee, Jr., and H. A. Stafford, both of Franklinton.

Orleans Parish: Drs. C. J. Brown, Philip Montelepre, Robert A. Robinson, Jr., Samuel A. Romano, C. J. Tripoli, Shirley C. Lyons, Manuel Gardberg, Howard R. Mahorner, George C. Miramon, William J. Rein, I. L. Robbins, John J. Archinard, Edward Matthews, Murrell Kaplan, Philip A. Boudreaux, James C. Cole, J. Holmes Smith (Deceased), W. Norton Voorhies, L. A. Monte, C. F. Bellone, Frank F. Gambino, Jr., W. G. Troescher, Jos. J. Ciolino, Lucien Delery, Edward B. Faget, Philip H. Jones, Jr., Benj. L. Newell, T. T. Batson, Hugh T. Beacham, Frank Chetta, F. C. Hava, David Hyman, J. Henry Larose, Jr., Lewis H. Levy, Warren L. Rosen, C. J. Wichser, Owen F. Agee, Louis L. Friedman, T. A. Jung, D. V. Longo, George A. Cronan, Joseph M. Hountha, T. F. Kirn, Joseph P. Palermo, Claude V. Perrier, Joel B. Gray, John Morgan Lyons, Harold A. Bloom, M. L. Michel, Rawley Penick, Jr., J. D. Russ, Willard Wirth, Woodward D. Beacham, Frank L. Cato, Jr., B. G. Efron, E. R. Guidrey, R. T. Liles, James L. Locascio, Frank L. Loria, Spencer B. McNair, Charles S. Healey, Peter Everett, Jr., H. B. Alsobrook, C. Gordon Johnson, J. W. A. Smith, Charles L. Bloom, Hymen Cohen, Lucien H. Landry, Alberto Prieto, J. D. Rives, A. F. Brock, P. J. Saleeby, E. A. Bertucci, R. S. Crichlow (Deceased), Esmond A. Fatter, F. Temple Brown, J. M. Davidson, B. Hochfelder, and J. M. Perret, all of New Orleans.

TREUTING NEW HEAD OF STATE BOARD OF HEALTH

Dr. Waldo Louis Treuting has been appointed to succeed Dr. David E. Brown as president of the State Board of Health. Dr. Treuting is a native of New Orleans and graduated from Tulane Medical School in 1934. After a year's internship at Charity Hospital he was appointed to the State Board of Health. For three years he was head of the division of epidemiology and preventive medicine. While serving the Board of Health he received his degree of master of public health in 1939 from Johns Hopkins University. His progress in the State Board of Health has been steadily upward and during the war he was, next to Dr. Brown, the highest ranking officer in the department.

Dr. Treuting is well trained for public health work. He should make an excellent director of the department. Governor Davis is to be congratulated on appointing a man who has made public health his life work and who is qualified in every respect as a well trained public health officer.

NEWS ITEMS

Dr. Howard Mahorner, assistant professor of clinical surgery, Tulane, addressed the Mobile Civic Forum recently on the subject of Socialized Medicine — the Phantom Approaches.

It has been announced by President Rufus C. Harris, of Tulane, that a grant of $35,000 for the purpose of studying the effect of climate on the circulatory system has been made to the university by the Life Insurance Medical Research Fund. This research work will be directed by Dr. George E. Burch, associate professor of clinical and experimental medicine.

A breakfast was given to the past vice-presidents of the American Medical Association during the recent meeting of the organization in San Francisco. Dr. L. H. South, of Louisville, and Dr. J. Watts Stovall, president of the Kentucky State Medical Association, were hosts. There are 21 surviving vice-presidents of the A. M. A. of whom two, Dr. Rudolph Matas and Dr. John H. Musser, reside in New Orleans.

Drs. George E. Burch, Travis Winsor and L. LeRoy Kimball, were one of three groups awarded a certificate of merit at the scientific exhibit of the A. M. A. The award is based on the originality of the investigation and the excellence of presentation.

At the quarterly meeting of the New Orleans chapters the Board of Directors of the Red Cross appointed the following medical advisory committee: Dr. Joseph A. Danna, Chairman, Dr. Val H. Fuchs and Dr. C. Walter Mattingly.

President W. B. Hatcher of the Louisiana State University announces an addition of 40 new faculty members to the university medical school plus 23 more members added to the staff of those who have returned from duty in military service since December 1.

Honorary memberships in the Brazilian Academy of Military Medicine were recently bestowed upon Major General Norman T. Kirk, The Surgeon General, and Colonel Leon L. Gardner, MC, Director of Army Medical Library.

INTERNATIONAL COLLEGE OF SURGEONS

The executive secretary of the International College of Surgeons announces that 17 operative surgical clinics will be held at the Detroit Assembly October 21-23. The convocation address will be delivered by Ex-Ambassador to Japan, Joseph C. Grew.

AMERICAN COLLEGE OF PHYSICIANS

The 28th annual session of this important organization will be held in Chicago April 28-May 2, 1947. Dr. David P. Barr, President of the College, is in charge of the program of general sessions and lectures. Dr. LeRoy H. Sloan is general chairman in charge of the program of hospital clinics and panels as well as of local arrangements.

SOUTHERN MEDICAL ASSOCIATION

According to the notice sent out by Mr. C. P. Loranz, Secretary-Manager of the Southern Medical Association, the Miami meeting of this organization will open at noon Monday, November 4 and will continue through Thursday afternoon, November 7. The general headquarters will be at the Municipal Auditorium in Bayfront Park. In this notice sent out by the secretary-manager, the various hotels are listed. It is suggested concerning hotel space that where the hotel of choice can not accommodate the applicant, reservations be made through the hotel committee.

After-meeting trips are being arranged for those who wish to go to Havana and/or Nassau.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

The National Society for the Prevention of Blindness will hold a three-day conference November 25-27 at the Hotel Pennsylvania, New York.

ARMY CONSULTANTS

The surgeon general of the army has announced that three New Orleans physicians are amongst the 74 civilian doctors appointed as consultants to the secretary of war. The three appointees are Dr. Champ Lyons, surgical consultant, Dr. Michael E. DeBakey, consultant in general surgery and military history consultant, both of the surgical department of the Tulane Medical School and Dr. Roy H. Turner, of the Tulane Department of Medicine, medical consultant.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending June 1 showed the following diseases in numbers greater than ten: Unclassified pneumonia 126, cancer 127, measles 106, pneumococcic pneumonia 59, pulmonary tuberculosis 32, amebiasis 24, malaria 24, mumps and rheumatic fever 18 each, scarlet fever 13, septic sore throat 12, influenza 11 and typhoid fever 10. There were three cases of poliomyelitis reported this week, two from Livingston and one from Iberville Parish. The number of pneumonias, unclassified and pneumococcic, is very remarkable for this time of the year. At the same time that the weekly morbidity report was sent out the monthly morbidity report for venereal diseases was distributed. For the month that ended May 31 there were listed 1971 cases of syphilis, 1710 of gonorrhea, 66 of chancroid and 20 of granuloma inguinale.

There was a truly remarkable fall in the number of reportable diseases for the week which closed June 8. The only disease which occurred in figures greater than ten was measles with 34 cases listed. However it can be noted that there were nine cases of poliomyelitis distributed throughout the state, no one parish having more than two cases. For the week which came to a close on June 15 there were sent to the State Board of Health reports of 117 cases of measles, 62 of pulmonary tuberculosis, 26 of malaria, 22 of mumps, 17 of septic sore throat, 13 of whooping cough and 11 of meningococcus meningitis. Only three cases of poliomyelitis were reported this week. For the week ending June 22 for the first time in many a week pulmonary tuberculosis led all other reportable diseases with 59 incidences followed by 55 of measles, 27 of mumps, 18 of whooping cough, 14 of septic sore throat and 13 of typhus fever. There were eight cases of poliomyelitis listed for this week, four of which originated in Orleans Parish; the others scattered throughout the state. So far this year there have been listed 58 cases of poliomyelitis, a figure well above that of last year.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that for the week ending June 15 there were 103 deaths in the City of New Orleans. These deaths were divided 71 white and 32 colored with seven of the deaths occurring in children under one year of age. This was a very marked reduction from the previous three year average. There was a fairly large increase in the number of deaths for the week which terminated June 22. Of the 137 deaths in New Orleans this week 89 were white, 48 colored and 12 infants under one year of age. The deaths had materially fallen in number for the week which came to a close June 29. The three year average for the corresponding week has been 160 but in this week only 115 people died in the city, of which 76 were white, 39 colored and five infants. The astounding low figures were maintained for the week which closed July 6. The 106 people who died in New Orleans were divided 67 white, 39 colored and 11 infants. The low mortality rate for the City of New Orleans for the past year has been truly remarkable.

LOUISIANA STATE BOARD OF MEDICAL EXAMINERS

Governor Davis has announced the reappointment of Dr. Roy B. Harrison, Dr. Leon J. Menville and Dr. Rhett McMahon to the State Board of Medical Examiners. These reappointments were made on recommendation of the House of Delegates of the Louisiana State Medical Society. These three men have proved their efficiency and their ability properly to carry out this somewhat difficult assignment.

CARROLL D. OVERTON

1902 - 1946

Dr. Carroll D. Overton, of Hammond, was shot and killed on July 1 by a former patient who then committed suicide. A tragedy such as this is one of the occupational hazards of medicine. Time and again doctors are murdered by insane patients who believe that their illness is dependent upon improper treatment.

Dr. Overton was a graduate of Tulane University Medical School and practiced medicine in Hammond after completing his internship.

BERT LANDON STINSON

1870 - 1946

Dr. Bert Landon Stinson, of Homer, died on July 8 of hemorrhage of the descending aorta. He was a graduate of Rush Medical College, 1900 and interned at Milwaukee Hospital, Milwaukee, Wisconsin in 1901. In 1927 he came to Amite, Louisiana, serving as Director of the Tangipahoa Parish Health Unit. From Amite he moved to New Iberia and in 1939 became Director of the Claiborne Parish Health Unit, which position he held until the time of his death.

WOMAN'S AUXILIARY

The Woman's Auxiliary to the Tangipahoa Parish Medical Society met with Mrs. DeLoach Thames on Thursday, July 11, for their regular monthly meeting, Mrs. Glen Scott, President, presiding. For the Program, reports were given on the state convention, recently held in Alexandria. A complete report was given of all activities by those who attended, Mrs. Glen Scott. Mrs. DeLoach Thames, and Mrs. M. C. Wiginton. Mrs. Wiginton gave a brief resumé of the report of the past president, Mrs. Paul Lacroix. These reports created so much enthusiasm among members that all decided they could not afford to miss the next state meeting.

Following the business session Mrs. Thames served very delightful refreshments.

In spite of the heat this was a most interesting meeting and it was very gratifying to have wives of the medical profession so congenial and enjoy such rich fellowship as was evident.

Two new members were added to the roll, Mrs. S. J. Dileo of Independence, and Mrs. R. H. Alldredge, associate member. Others present were Mrs. Glen T. Scott and Mrs. Collins Lipscomb of Ponchatoula, Mrs. S. J. Cali, Mrs. C. D. Alessi, Mrs. M. C. Wiginton.

Other auxiliaries disband during the summer months, but Tangipahoa Parish, so newly organized, thought it wise to have regular meetings, and although these meetings will be purely social they promise to be very entertaining and interesting. In the fall when other auxiliaries begin to function, Tangipahoa Parish Auxiliary will begin regular activities.

BOOK REVIEWS

Normal Lives for the Disabled: By Edna Yost and Lillian M. Gilbreth. New York: Macmillan Company, 1945. Pp. 298. Price, $2.50.

This admirable volume is published especially for those who are mentally or physically impaired because, with relatively few exceptions, handicapped persons can attain economic independence and a normal or satisfactory social situation. It tells them how to go about it and is most helpful for their families and friends — for everyone who wants intelligently to take part in helping the disabled achieve useful, happy lives.

Every handicapped individual-civilian or veteran can and should initiate reconstructive thinking and efforts to help himself as early as possible. From years of experience, the authors know that everyone, when real need arises, has a superiority in one line or another which can help compensate for physical or psychologic loss even when the defect is a severe one. They have seen countless men and women — not just those with unusual talents, gifts or skills — conquer their defects, find suitable places in business or industry, and enjoy themselves, their homes and their friends.

Here is a comprehensive, practical and intelligently presented survey of what has been and is now being done to help impaired persons rehabilitate themselves. It reflects the views and experiences of specialized teachers, industrialists, psychiatrists and psychologists, as well as of physically or psychologically injured men and women themselves who have successfully solved their difficulties. It also contains valuable material on vocational problems as they must be met by the variants, and excellent descriptions of these men and women in an amazing variety of jobs in the United States today.

Though small in size this manual possesses qualities and values which are superlative. Its service offerings and standards are high. Its material is designed to keep pace with new human needs and demands. To reiterate, it engagingly expresses the authors' convictions of the self-respect and happiness which handicapped men and women can attain.

C. P. May, M. D.

The Intervertebral Disc: With Special Reference to Rupture of the Annulus Fibrosus with Herniation of the Nucleus Pulposus: By F. Keith Bradford, M. D. and R. Glen Spurling, M. D. Springfield, Ill., Charles C. Thomas, 1945. Pp. 192, 70 illus. Price $4.00.

This second edition of a truly fine monograph brings one up to date on the important subject of the intervertebral disc. Importance is stressed in the early part of the book on the fundamental anatomy, physiology and pathology of the subject. The relation of these basic facts and their correlation with the clinical syndrome is adequately expressed in the second portion of the book.

The localization and differential diagnosis of the ruptured annulus fibrosis is made clear by means of tables and drawings. The procedure following a diagnosis of first following conservative measures, and only resorting to surgical removal of the disc when these fail is sound medicine in a comparatively new syndrome. The authors do not state which spines should be fused, although they discuss the fact that this procedure should be followed in certain cases. However, in their case reports no spines are fused.

Although 13 case reports are given, only one case of mistaken diagnosis is mentioned. In the differential diagnosis little attention is given to mistakes in diagnosis.

This monograph is adequately indexed, profusely and well illustrated, and is recommended for its research, clarity and context to all physicians whose patients complain of persistent backache.

Fred S. Marshall, M. D.

Injury and Death Under Workmen's Compensation Laws: By Samuel B. Horowitz. Boston, Wright & Potter Printing Co., 1944. Pp. 846. Price $6.00.

Compensation acts in the United States are now of age. All over the country there are thousands of men and women injured every year in industries and existing acts are a boon to them and their dependents. Annually 2,000,000 workers become casualties; 18,000 die; 100,000 are maimed for life; and the rest recover after varying periods of time. About $200,000,000 a year reach the hands of workers, their dependents, and those who furnish medical and hospital care.

Although the basic theory and principles of workmen's compensation are relatively simple, their practical application involves a tremendous number and variety of important administrative details and requires a high degree of talent and alert, diligent effort. And, too, compensation laws are put on the books for the benefit of workers, not employers or insurance companies.

In the sections on Personal Injuries and By Accident, there are many important observations of which the following are interesting examples: (1) Nervous shock causing incapacity for work is as much a personal injury by accident as a broken limb or other physical injury. When you have that which requires treatment of the body, it means that a portion of the body (visible or invisible does not matter) is in a state of ill health. (2) Traumatic neuroses following physical injuries are almost universally compensated, even though financial, marital and other worries play a part. So, too, are many injuries to nerves, even by invisible but poisonous coal tar gas. (3) Aggravations of pre-existing diseases or defects are as compensable, in most states, as an original or new injury. (4) Employers take workmen "as is," this is, without any warranty as to any previous state of health, known or unknown. Hence effects of injuries, intensified by pre-existing, underlying or latent disease, are very often compensable. To help cripples obtain jobs and to assist in the payment of such aggravations, the majority of states now have second-injury or similar funds. (5) Where a man sustains a nervous shock, producing physiological injury, not a mere emotional impulse, he meets with an accident. Physical impact, while common-law cases, is not necessary in compensation cases. If during a fright he falls and injures himself, or "drops dead," the injury is by accident.

Under Incapacity it is stated that one who has only a partial physical disability may obtain total incapacity payments on proof that he was unable to market his remaining capacity for work. And for permanent, total disability the disability must be permanent, that is, one that will continue for life or indefinitely. The fact that it is merely conceivable, but not probable, that improvement will take place, does not prevent a finding that the disability is permanent.

Throughout the volume there are numerous pertinent citations of court decisions and there is an excellent index and a bibliography.

Except for certain medical aspects this book is more suitable for a legal review. But it is a rare combination of research and synthesis, a work to be deeply pondered. It offers no short cuts or magic solutions but it contains the clearest discussion of its subject matter that I have ever encountered. Its use should bring about more accord in controversial or litigated issues.

C. P. May, M. D.

The Venous Pulse and Its Graphic Recording: By Franz M. Groedel, M. D. New York, Brooklyn Medical Press, 1946. Pp. 216. Price $5.50.

The author and his colleagues have been interested in the study of the venous pulse for many years. This small monograph presents some of their methods and results as well as review of the pertinent literature. Dr. Groedel correctly emphasizes the point of view that we cannot complete our knowledge of the circulation until we study the venous blood stream with the same interest and enthusiasm as we do the arterial blood stream. The author candidly admits that the study of the venous side of the circulation is difficult because of the presence of many variables. The development of modern methods of recording, however, has made it desirable to attack the problem again.

The monograph is divided into three sections. The major part is devoted to a detailed discussion of the phlebogram, the graphic representation of the venous pulse. The other two sections are concerned with the pneumo-cardiogram and the esophago-cardiogram, respectively.

The author uses an oscillograph and galvanometer system similar to that found in the electrocardiograph in conjunction with a high frequency microphone. Two types of waves are distinguished in the phlebogram: (1) the venous tidal waves (a, c, v, etc.) reflect the pressure-suction effect of the heart upon the blood flow in the larger veins and are therefore primarily manifestations of fluctuations in intrathoracic pressure; (2) the concussion waves due to the concussion of the blood column in the large veins caused by the opening and closing and contraction of the heart chambers. Although this method of analysis represents a distinct advance, it is apparent that much more work needs to be done to determine the origin and significance of the various waves found before the method can be used in diagnosis.

The author uses a gas mask connected to the microphone for the determination of the pneumo-cardiogram. Likewise, an esophago-cardiogram is obtained by connecting an Einhorn cardia-dilator with the microphone. These records are correlated with the phlebogram. Such a comparison indicates that when proper technics are used, the three types of records represent nearly identical tracings reflecting the mechanical events that are taking place in the heart and the intrathoracic pressure changes due to systole and diastole of the various heart chambers and to the inflow and outflow of blood from the thorax.

This monograph merits the serious consideration of those who are interested in the circulatory system. Phlebography was enthusiastically taken up after the first World War and almost as enthusiastically dropped when it became apparent that technical difficulties made it difficult to obtain good phlebograms. The strides made in electrocardiographic diagnosis created the feeling that most of the information given by the phlebogram could be obtained much more simply and clearly with the electrocardiogram. While this is true of many clinical conditions, it is now apparent that other methods must often be used to supplement the study of the electrocardiogram if we are to diagnose successfully various clinical conditions. Phlebography should prove of great value in this connection. The methods are sufficiently developed at the present time and enough is known of the significance of the various waves to justify its use in clinical examination.

H. S. Mayerson, Ph. D.

Medicine in Industry: By Bernhard J. Stern, Ph. D. New York: The Commonwealth Fund, 1946. Pp. 209, tables 15. Price $1.50.

This small book is another of the several studies of the New York Academy of Medicine Committee on Medicine and the Changing Order.

In this monograph, Dr. Stern, Columbia and Yale University sociologist, surveys the past accomplishments in industrial medicine and draws from the data suggestions for future development. The data used in the fourteen tables are derived from dependable sources, as from the U. S. Department of Commerce Bureau of the Census, the U. S. Department of Labor, the U. S. Public Health Service, Senate Subcommittee Reports of the War Manpower Commission, and so forth. Certain references used in the body of the text, however, do not appear to have been so carefully chosen. For example, on page 162, the author contents himself with 1939 source material to arrive at such deductions as the frequency of laboratory examinations as a part of the physical examination given by industrial physicians. Are these findings representative of 1946, some seven years later, when organized labor, with better understanding of the purposes of industrial medicine, complains less about factual laboratory data?

Probably the best written of the seven chapters of the book is Chapter II, Social and Legislative Backgrounds. This chapter, with Chapter VII, Preventive Services, offers good material for reading assignment to medical students during their work in industrial medicine and hygiene.

Ralph H. Heeren, M. D.

Group Psychotherapy: *Theory and Practice*: By J. W. Klapman. New York, Grune & Stratton, 1946. Pp. 344. Price, $4.00.

Group psychotherapy has been practiced, after a fashion, since the advent of mankind. But, as a selfconscious discipline with an attempted appraisal of causes and effects group psychotherapy is of relatively recent origin.

Its concepts, theories and technics are still in an incipient stage, but recent, urgent stresses in world affairs have served to accelerate and intensify the need for it. Because of increasing numbers of military and civilian psychiatric casualties and enlarged demands for psychiatric services in general, group-psychotherapy has suddenly been called upon to fill a role of unprecedented proportions.

While this art is comparatively new there has been time and opportunity for certain exact formulations. It is time for a summation of current usages to indicate some guiding, even though tentative, principles for those who contemplate employing group-psychotherapy.

It is believed that the important contribution group-therapy makes to the healing arts constitutes more nearly a living-through process in relation to the social setting than any other form of psychotherapy.

The range and variety of groups which may be treated by group-psychotherapy are seemingly endless. If it is borne in mind that the principles and dynamics of group-psychotherapy are essentially the same for all groups, though the modes of application may vary, few difficulties need arise in attempts to treat new, untried groups.

Group-psychotherapy is more than a make-shift in the service of economy. It extends the service of each practitioner to more patients than can be individually treated. It has the unique advantage of being able to utilize the collective will, to mobilize the contagion of the group, for therapeutic gains. It demonstrates that many persons in need of treatment, who are withholding during individual interviews, will force and discuss their psychic difficulties and traumas when they find they are not alone in their troubles. The profound importance and value of this excellent publication goes without saying.

C. P. May, M. D.

Suggestion and Hypnosis Made Practical: By Samuel Kahn, M. D., Ph. D. Boston, Meador Publishing Co., 1945. Pp. 200. Price, $3.00.

Suggestion is the most powerful force man uses on man. Success in human relationships is certain for those who understand and use it. It is universal in its influence and is a determining factor in getting what one wants.

The therapeutic value of suggestion in the treatment of disease has been known to mankind since the earliest stages of recorded history and it was through the efforts of Mesmer, about 1770, that a real beginning was made in what is now known as scientific psychotherapy.

Transference of ideas from one person to another is cosmic; it is this that makes possible the great effectiveness of suggestion. Ideas are contagious because of the mechanism of suggestion. Thoughts — whether they are noble or evil — if repeated often enough and with conviction, become forces that influence actions.

Generally, a patient prefers to have his problems solved by the doctor. In suggestion and hypnosis, the operator does the thinking. In analysis, the patient thinks for himself and solves his own problems under the physician's guidance.

Hypnosis, alone, cannot cure. It affords the patient no insight into the darkness of his mind, and at best can offer him only an escape from reality. Suggestion and hypnosis may undermine the individuality of the patient and make him dependent on the operator. Analysis, on the other hand, will restore his individuality, giving him strength and courage through understanding and knowledge. It is only when the patient recognizes his internal adversary that he is able to cope with it. What has been a hidden conflict with the inner enemy becomes an open struggle. In no other way can a lasting cure be achieved. I see eye to eye with the author in these views.

Chapter 4, Auto-Suggestion, is interesting and valuable. Auto-suggestion, the use of suggestion on oneself, is a form of self-education, because the individual uses it to influence himself. With it, he talks himself into believing ideas in order to achieve specific goals. Auto suggestion is a technic for educating, training, and influencing oneself.

Powerful results can be built up by auto-suggestion. There is a "summation of results," or "habit formation." The prevention of mental conflict by re-education and autognosis, and the avoiding of negative auto-suggestion, can be successfully accomplished through repeated suggestion on oneself.

While this book treats suggestion in general there are in it chapters on special phases of using suggestion as in medicine, business, rumor-propaganda. Its use calls for realistic, inductive and creative thinking. Its author has had broad training and experience. It can help any intelligent person attain greater mental efficiency and a fuller richer life.

C. P. May, M. D.

Modern Clinical Syphilology: By John H. Stokes, M. D., Herman Beerman, M. D., Sc. D. (Med.) and Norman R. Ingraham, Jr., M. D. 3d ed. reset. Philadelphia, W. B. Saunders Co. 1944. Pp. 1332. Price, $10.00.

When one undertakes the task of reviewing the third edition of Stokes' Modern Clinical Syphilology he soon discovers that he is confronted with a task which challenges his ability to concentrate on the problem of syphilis, with the many ramifications of the disease. This is not a criticism of the material in the book since indeed Stokes' Modern Clinical Syphilology is an encyclopedia of syphilis. The phrase "You can look it up in Stokes" is as true of this edition as it was of former ones. The serious student of syphilis can benefit especially from referring to this volume. On the other hand, the busy practitioner who might want to look up a specific problem, such as the exact definition of Grade 1 spinal fluid, would not readily find the answer to his inquiry because he would be inclined to seek information in the chapter on Central Nervous System Syphilis, rather than in the chapter dealing with diagnostic procedures. There seems to be no clear-cut advice to the practitioner concerning the advisability of continuing to treat sero-resistant cases, and what may be expected from further treatment.

In Figure 438 concerning the prevention and the treatment of relapse, this statement is made: "Turn to 606 if 914 has failed, or use it from the start." There are other bits of advice in this figure which leads one to believe that this segment of the book has not been recently revised to consider such therapeutic agents as mapharsen or one of the other newer phenarsine preparations.

In the section on differential diagnosis between secondary syphilis and other skin ailments, one wonders why Stokes has not specified finding treponema pallidum by means of darkfield examiation of syphilitic lesions as *the* diagnostic distinction between papular or annular manifestations of syphilis and the other skin diseases being considered. Only in Figure 314 is the finding of T. P. mentioned as a diagnostic feature (as distinguished from lesions of leprosy).

The last two chapters are timely and as up to date as could be managed. It is surprising to find the relationship between military and public health syphilis handled as completely as it is in chapter 33, when one remembers that it must have been added to the book late in its production. But the ultimate in getting material to press is achieved in the last chapter in which Stokes discusses penicillin and gives a preview of its use in the treatment of syphilis. If some of his statements should not prove to be entirely accurate, one should ask the question "Could anyone have judged better except in retrospection?".

On the whole this revised edition of Stokes is a most valuable book, perhaps the world's outstanding reference book of syphilology.

Owen F. Agee. M. D.

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THE PHYSICIAN AS A WORLD CITIZEN*

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On important university occasions such as this, a university looks at its work and its relation to the society of which it is a vigorous, indeed essential, expression. Its greatest works, of course, are its scholarship, its science, its humane letters, but above all, its human products—the young men and women who have had the privilege of associating with great minds and skillful hands, the devoted men who have carried on the unbroken humanistic tradition of university learning and culture for more than a thousand years.

It is well to remind ourselves in this turbulent century that universities are among man's most durable institutions. Most of them have survived all the vicissitudes of life and of states. A. Lawrence Lowell remarked a few years ago that those which have disappeared did not die from attacks from without but died from within. Its strength and durability grow out of the university's vital intellectual ferment, its identity with the community and its needs, and at the same time its detachment and objectivity, its integrity of thought and purpose, and its selflessness. But most of all it is constantly renewed and renourished by the influx of new students with fresh points of view and inquiring minds.

Medicine has been part of this great tradition and has enriched all other university disciplines through its interest in health and disease, in human growth and development (intellectual and spiritual as well as physical), its interest in the natural sciences, the social sciences, and the humanities. It is well known that physicians of ancient Egypt and Greece, who laid the foundations for modern scientific medicine and its moral and ethical codes, were also among the first great naturalists and humanists.

In our time, however, the roots of medicine in the fundamental disciplines of the university seem to be farther and farther removed from the every day work of the clinic by the division of labor in medical teaching and practice among numerous basic and clinical specialties. This has taken place despite the recognition by medical schools for the importance of these disciplines as a preparation for medicine. Yet on closer examination one can see that the link between the clinic and the university in all of its aspects is very close indeed. Everyone is aware, for example, that the revolutionary advances in nuclear physics and chemistry have important implications for medical and biological studies generally. Wartime advances in medical science, great as these have been, will be insignificant compared with what lies ahead in our lifetime for all branches of science, including medicine. Today medicine is more firmly than ever rooted in its sister disciplines of the university, not excluding the law and the other social sciences. A leader in legal medicine recently wrote: "Law will aid men of medicine and of science to extend the social effects of their discoveries, to eradicate certain maladies, to help cure the widespread disturbances of physical and mental health which now spring from socio-economic maladjustments. Between law and science the whole fabric of society may be spun anew."

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*Commencement address delivered at The Tulane University of Louisiana, School of Medicine, on May 15, 1946. A portion of this paper is paraphrased from the writer's monograph on "Medical Education and the Changing Order," published by the Commonwealth Fund under the auspices of the New York Acamemy of Medicine's Committee on Medicine and the Changing Order.

Increasingly medicine has focused its attention on man as a social organism. We have a growing realization that, while there is still much to be learned about the physical aspects of the life of man, the great contributions of the future will come from the exploration of man's relationship to the society which makes him what he is and which he constantly attempts to mold to his desires. Justice Holmes put it more succinctly when he wrote: "Man, whether he realizes it or not, is always fighting for the kind of world he wants." In man's constant struggle to find happiness in an ever-changing technological world lies the challenge for any student of the future. Here if anywhere, in the ever new demands upon both society and the individual, will he find the answers to the many medical riddles that confront us today. Here perhaps he may begin to understand and help the suffering human beings in whom we can find no apparent physical reasons for suffering.

The study of medicine, therefore, must embrace the study of life in all its aspects, physical, psychological, and social. It is not suggested that the physician must be an expert in sociology, economics, law, business, personnel management and in countless other fields. It *is* suggested, however, that he should have a clear and comprehensive appreciation of the place of these aspects of society in the life of the individual. He should realize, for example, that preventive medicine is being practiced at a most effective level by the forward-looking businessman who takes an active interest in such things as employee nutrition, health services, housing, safety and recreation, as well as the cause of absenteeism, juvenile deliquency and other social problems. Likewise, the physician should recognize as an ally a government which attempts to remedy social, economic and industrial deficiencies and abuses. Such efforts will play a most important part in the doctor's struggle against those untoward environmental influences which make for occupational disease, maladjustment and degenerative changes in the individual.

"Society is indeed a contract . . . But the state ought not to be considered as nothing better than a partnership . . . to be taken up for a little temporary interest, and to be dissolved by the fancy of the parties . . . it is a partnership in all science; a partnership in every virtue, and in all perfection. And as the ends of such a partnership cannot be obtained in many generations, it becomes a partnership not only between those who are living, but between those who are living, those who are dead, and those who are to be born."—Edmund Burke.

Any well-educated and experienced physician realizes that the professional services of lawyers, ministers, teachers, social workers and personnel counselors are essential in supporting and helping the individual to make a successful adjustment to the conditions of a complex modern life. Imagine a society without one of these essential professional components. One sees at once that their friendly personal services, frequently offered without thought of financial gain, are the very keystones upon which society is built. This is something that can never he mechanized. Real professional service may have its technics, but, it cannot be a technology: It takes a man, not a machine, to understand a man. Useful as machines may be in making observations and recording man's reactions and actions, the information they provide is useless unless we have someone able to interpret it and make judgments on the basis of wisdom born of experience. Essentially, this is the function of the professional man, whether he be a physician, teacher, minister or lawyer. Out of the wisdom of his life's experience he must make decisions and recommendations likely to help those who consult him. It is simply a case of the skilled and, therefore, the strong helping those who are relatively less fortunate. Knowledge and skill in the use of knowledge are power; they are the source of the authority and influence of professional men. The use of such power can be entrusted only to men and women of intelligence, character, and fidelity, for in them is the future wisdom of the race.

Medical education aims to produce physicians who are not merely skillful, but wise as well in the application of the art and science of medicine. To be wise, the physician must understand the physical and social setting in which his patients live. As a physician and a highly privileged citizen, he must encourage and participate in every community effort to improve living and working conditions for everyone. Thus he fulfills his obligation to attack disease and suffering at their sources and to remove as far as possible every aggravating cause of human disability.

The interest and enthusiasm of men in professional and business life are essential for community progress; but civic pride, vigor and leadership are derived from every phase of community life. The small and big businessman, banker, lawyer, engineer, druggist, doctor, minister, teacher, members of women's organizations, labor unions, chambers of commerce and governmental agencies, all contribute leadership to the public welfare. The doctor enjoys the respect of the community because of his learning and skill. He represents the viewpoint and detachment of science in the discussion of complicated health and community problems. If he can add the qualities of integrity, tolerance, fairness, kindness and friendliness, his influence for the good of the community will be beyond measure. In some backward areas, in fact, he may be the only person who exercises any influence for constructive leadership.

Most physicians who are attracted to community life are the kind of people who do not allow the technical and scientific character of their training to obscure their natural human approach to patients and fellow citizens. As the community demands more preventive medicine and gives greater and more serious attention to such evidences of social deficiency as poverty, unemployment, industrial strife, malnutrition, high rates of juvenile delinquency and illiteracy, the doctor will come to occupy an even more important place of social responsibility. Therefore, the doctor must prepare himself to fill this broader role of leadership. He must equip himself with a thorough understanding of the social, industrial and economic patterns of which he is a functioning part.

Now I should like to focus your attention for a moment upon a university's interest in the one great question which is of first importance for any thinking person today. This is the crisis between technology and the higher values in life and living for which men have been striving for centuries. The question is whether man has sufficient wisdom, humility, and social consciousness to use his newly-found powers for the constructive purposes of all mankind. Free men have proved that democracy, and our republican system of self-government particularly, has the inherent strength to be victorious in a world at war; they have yet to prove that they can win a durable peace. Never before has the intellectual, spiritual, and moral life of man faced a test of such a portent for good or evil. Medicine with its age-old concern for the sick, the poor as well as the rich, the weak as well as the strong, has been an influence for good surpassed only by the moral precepts of religion. The services of medicine, like those of religion, have been largely personal. There will always be need for personal services, but medicine of the future, if it is to progress as a social as well as a biological science, must broaden its outlook and adjust its educational program accordingly. Medicine is coming of age as a social science.

Thinking and planning for democracy must be bold and dynamic, drawing not only upon the talents of individuals, but also upon the social discipline of the group. At times, unfortunately, our system falters. We have great difficulty in distributing evenly the goods and services, including medical services, that our expanding technology pours forth in ever greater abundance. Our social process lacks stability which, perhaps, is but a sign of growth and change. This should not be too alarming. But, when the system becomes so unstable that it upsets our relations with our neighbors and when we lack the self-discipline and social controls to resolve our differences rationally and peaceably, then just to this extent has education failed at one of its primary tasks.

College graduates generally fail to take an interest in legislation, state or federal, even when it intimately affects their own fields of business or professional activity. We have always maintained that training for leadership is a major function of a university. If this is true, then we are failing in our purpose when education does not develop in the individual a social and political consciousness to the same degree that it trains him for professional and vocational proficiency. If this is true, may it not also be that here is a significant reason for disharmony between government, that is, social controls within the framework of law, and the citizen who still believes that "that government is best which governs least"? Are our schools, at all levels, sufficiently conscious of their obligation to educate for responsible citizenship? This, I believe, is the most important question we must resolve if our Republic is to survive in the critical years through which we are passing.

But there is a larger question, one that concerns every American, whether he is a college man or not. It is: What has happened to our greatest national asset, the native American spirit; the selfless, Christian spirit that founded this great nation, that unified and preserved it from internal division, that industrialized a continent and that, twice in this century, joined with and led the peace-loving peoples of the earth in conquering tyranny and preserving our right to live as free men? To live for what? For the good life for all men everywhere? Or for the giddy life of getting and spending in which human values are overwhelmed and smothered by the petty value of material things? If the latter is our answer to a starved and war-torn world, if we demand an island of selfish American plenty within a world of want, then we are a dying nation with no hope of realizing our true inheritance of greatness. We are left, it seems to me, with only one choice; we must elect to bring the machine under social control; we must decide that economics should be made to serve the good life for everyone rather than merely that of a favored few; we must recognize that it takes a man, not an adding machine, to understand a man.

Time is short and apparently getting rapidly shorter. Universities must join hands with enlightened men everywhere, in the church, in business, industry, and labor, in the professions and in statecraft, to strive for the realization of our native American spirit and for greatness in the "brave new world" in which we live.

Universities must meet their obligation to the society from which they derive their right to exist. Today universities are meeting this obligation by adjusting their educational programs to emphasize, in equal measure, the development of social and political consciousness and training for proficiency in professional and technical skills. As medicine assumes its full stature as a social science, we may confidently expect the physician of the future to take his accustomed seat in the councils of community and state as a responsible and constructive citizen. To him the people may rightly look for leadership.

Medicine, like the peace, is indivisible. Its only concern is to advance its understanding of man, in health and disease, wherever on the face of this tortured planet he happens to live and to bring him as much relief from pain and disease as the knowledge and skills of the physician make possible.

Now may I congratulate this splendid

class upon its graduation from this distinguished and venerable University. Its pride in you, I trust, will equal your pride in being alumni of the Tulane University of Louisiana. This is an open door to the society of learned men everywhere in the world.

Thus today you as physicians have become world citizens, for learning, science, and medicine know no national boundaries. This is your heritage from the ages which I know you will cherish always. It is the gift of your University, a gift which I am sure you have richly earned.

Finally, speaking now as a physician, may I prescribe (with the permission of my friend, Dean Lapham, since I do not have a license to practice in Louisiana) a capsule of distilled wisdom for each of you. It is to be taken at your discretion as required by any circumstances in which you may find yourself. This prescription was first compounded by a great man of letters, Robert Walpole, who gave this advice to the young and I give it to you, the class of 1946: "Always good sense, good manners, good humor, and good faith." And may I add faith in yourselves, your country, your families, your fellowmen, and your God.

MIXED TUMORS OF THE PAROTID GLAND

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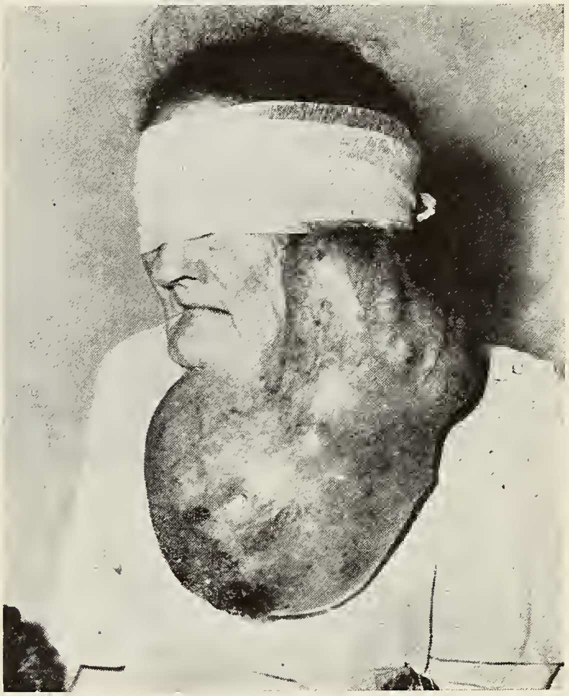
Mixed tumors of the parotid gland are of complex structure. Microscopic examination usually reveals them to be of epithelial elements and mesoblastic tissues, chiefly cartilage, mucous tissue and cellular connective tissue. Any one of these elements may predominate, but usually all cell types are represented.[1]

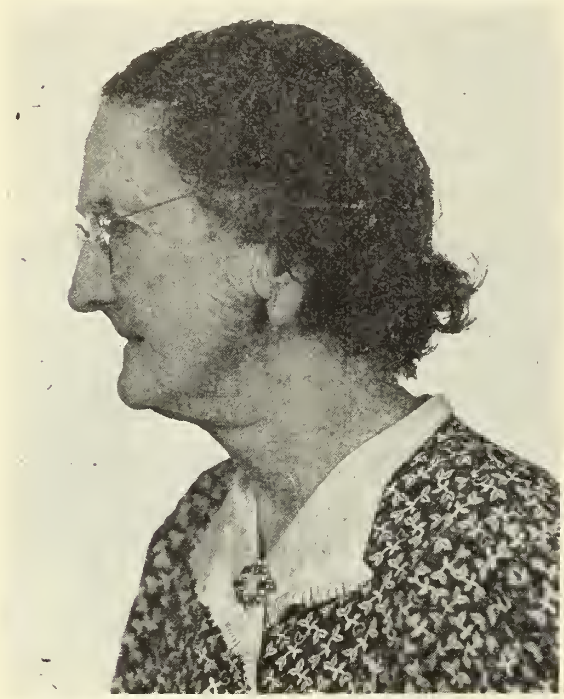
These tumors may be found attached or lying in the substance of the gland and are noted on physical examination when they have attained a size sufficient to appear as nodules or masses in the area of the parotid gland or below or behind the ear and some seem to involve the aural cartilages. The predominating element of the tumor determines its consistency. Most of these tumors are firm and hard, but growths predominately cellular or glandular may be soft. On dissection the smaller tumors are usually found to be encapsulated, but in the larger tumors fusion with the connective tissue of the gland is noted.

These tumors are not rare. Stein and Geschicter[1] report an incident of one of every 208 persons out of 50,000 surgical patients seen; 17.4 per cent of this series were considered to be malignant. Boyd[10] reports 103 cases from the Mayo Clinic between 1912 and 1917 and 69 cases from Memorial Hospital, New York, between 1918 and 1921. Martin[2] reported on 65,351 patients admitted to Barnard Free Skin and Cancer Hospital during a 24-year period and stated that 70 exhibited mixed tumors, of which 34 were simple and 36 malignant mixed tumors. Martin's reported incidence is, I believe, an actual low because most of these tumors are seen and treated by general practitioners and general surgeons. Patey[3] reports that benign tumors are more common than malignant in a ratio of four to three.



Patient Before Operation

The incidence is higher in the white race (80.1 per cent) than in the negro race (19.9 per cent) in a ratio of four to one.[4] There is an almost even division as to sex and size of body.



Postoperative Appearance of Patient

Mixed tumors have been found in persons ranging in age from 11 months to 73 years.[1] The age of appearance has no bearing on the benignancy or malignancy.[7] The most frequent age of appearance, however, is in the third and fourth decade.[8]

Ewing[1] summarizes our knowledge of their origin as follows:

1. The endothelial origin has been disproved.
2. No single source of the mixed tumors meets all requirements.
3. The derivation of mucous tissue and cartilage from gland epithelium has been satisfactorily proved and there is no necessity of including in the originating tissue any cartilagenous structures.

Ewing therefore classifies these tumors under the heading of epithelial tumors.

Mixed tumors follow a fairly common clinical course. They may appear at any age, but most often in the third decade as a firm, hard, painless nodule in the parotid area, below or behind the ear or seemingly as part of the aural cartilage. There is a gradual increase in size over a period of many years. These tumors usually double their size about every five years or so.[7] Tom Spec Jones of Baton Rouge had a case of a 67-year-old white female with a mixed tumor of the parotid gland of 51 years' duration which was removed surgically. The tumor "was more than a foot long and measures more than 21 inches in circumference." The pathologist reported the weight of this tumor as nine pounds. The largest mixed tumor on record was one reported by Canterell in the Scottish Medical and Surgical Journal in 1907 which is said to have weighed 26 pounds.

Benign growths are characterized by their lack of pain and slow steady enlargement. Malignant growths enlarge more rapidly and patients with malignant tumors complain of severe pain radiating to the face, neck, jaw, ear and back of the ear, which pain is probably the result of pressure on the fifth nerve.[11] Ulceration is uncommon until growth is sufficient to interfere with circulation.

It is fairly generally conceded that malignancy is not a primary clinical entity, but rather a secondary change in these tumors. Boyd [10] and McFarland[7] report that excision fails in about 25-30 per cent of cases and Boyd[10] that recurrences are more common when the tumor is removed while small. Ewing[1] states that surgical removal is often followed by recurrences of increasing malignancy. Montanus[12] is of the opinion that there are no criteria which will enable one safely to predict for mixed tumors a benign course, local recurrence or distant metastasis.

Patients with benign mixed tumors live indefinitely; patients with malignant disease rarely live over two years after operation.

The present methods of treatment are: (1) A laissez-faire attitude; (2) surgical excision; (3) irradiation; (4) a combination of irradiation and surgery. The status of irradiation at present is a matter of controversy, but apparently, except for palliation, it has not been effective.[2] However, the effect of preoperative irradiation seems to be beneficial to the surgeon in that it renders the capsule of the tumor tougher and less liable to rupture during operation.[3]

The important surgical-anatomical fact to remember is that the facial nerve emerges through the stylomastoid foramen and runs forward in the substance of the parotid gland. The nerve lies one inch deep to the midpoint of the anterior border of the mastoid process and a line drawn horizontally forward from that point to the posterior border of the ascending ramus of the mandible. As this nerve may be injured in the removal of benign tumors it is advisable to explain to the patient and to the family, preoperatively, that following surgery there may be great facial disfigurement as a result of section or paralysis of this nerve with resultant dribbling from the mouth, affected speech, impaired mastication, a blank stare to express all emotions and the weeping eye that cannot be closed.

The indications for surgery are: (1) The discomfort of the patient; (2) suspected or known malignancy of the tumor.

The predominant type of recurrence is local, but it may be to distant organs.

The differential diagnosis of these tumors is from: (1) Epidemic or infectious parotitis; (2) benign or malignant lymphadenopathy; (3) salivary calculus; (4) actinomycosis; (5) tumors of the jaw and antrum; (6) peritonsillar and buccal abcess.

CONCLUSION

It would appear that one should be in no hurry to treat these tumors, but should subject the patient to as long a period of observation as necessary to determine the rate of growth and possible malignancy of the tumor, more especially since malignant degeneration is many times more common following excision.

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DISCUSSION

Dr. Tom Spec Jones (Baton Rouge): Dr. Martin has covered this subject in a thorough and interesting manner, and I have only one or two points of interest that may be added.

In my service we have had three of these massive mixed tumors of the parotid gland. All three were in white females past sixty years of age and all were on the left side. They weighed nine, six and four pounds respectively. It is my belief that tumors of the parotid are not common. The incidence given by many authorities varies within very wide limits. They are of the mixed-cell type, containing much connective tissue, and the cells are often imprisoned in cartilaginous and bony tissue, which is an effort at restraint by nature. When compressed between the fingers it has the feel of a tight cyst, but upon opening the capsule you find it composed of a mass of solid fibrous material.

There is usually no urgent need for haste in operating, and every effort should be made to determine, preoperatively, if there be any suggestion of maligancy, for of course that would determine the prognosis. For instance, a tumor which has been present, unchanged except for its gradual increase in size for a period of 40 or 50 years, is certainly not malignant.

There are four important facts to be kept in mind when you have decided upon operation.

1. Always warn the patient and family of the danger of injury to the facial nerve and its consequences.
2. Tumors of the parotid are not prone to become malignant, but, when they are, recurrence is quite likely to take place.
3. Radium and x-ray do not seem to have very much effect on the malignant gland.
4. Be careful not to injure the nerve. Remember, normally, it passes through the substance of the gland.

As a rule those tumors are painless and symptomless, except for the size and weight. In the largest of our series (nine pounds) most of the weight was carried by an attachment of the capsule to the masseter and mastoid branch of the sterno-mastoid muscles. In addition, this lady had learned to support some of the weight by allowing it to rest on the shoulder, where she carried a permanent scar on the skin from the many years of pressure.

Because of the enormous size of those massive tumors and the consequent displacement and deformity of the ear, scalp and adjacent skin and soft parts, it is difficult to estimate the amount of skin to be removed. Make allowances for the postoperative atrophy of the skin and be certain not to remove too much.

SOME COMMON ERRORS IN FRACTURE TREATMENT*

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The doctor who handles fractures can in some ways be compared to the driver of the automobile. The driver accepts the responsibility of guiding his automobile safely over the road to a successful destination; the doctor accepts the responsibility of guiding the fracture of his patient safely over a course of healing to a successful termination, namely the restoration of function in the injured part. Both the doctor and the driver usually realize their responsibilities and feel able to cope with them but despite their good intentions, unforseen accidents occasionally arise which always damage the automobile and sometimes injure the driver. It is only by a careful analysis of each individual accident that a disaster of the same type can be avoided in the future and the accident rate in general lowered.

A few of the common causes in fracture treatment are: (1) failure to trim adequately the cast in the palm of the hand in the treatment of Colles' fracture; (2) failure to include the elbow and wrist joints in the x-ray of any fracture of the shaft of the ulna; (3) failure to reduce accurately epiphyseal fractures about the elbow; (4) failure to reduce supracondylar fractures of the humerus as soon as possible after the injury; and (5) conservative treatment of long oblique or spiral fractures of the lower third of the shaft of the tibia.

Following reduction of the Colles' fracture, a plaster cast is usually applied either from the axilla to the metacarpophalangeal joints on the back of the hand or from the elbow to the metacarpophalangeal joints on the back of the hand. If care is not taken to trim the cast back to the palmar crease in the palm of the hand, a complete range of finger motion is difficult or impossible, and the patient many times develops permanent stiffness of the fingers from capsular contraction which occurs during the period of immobilization. Old people are particularly subject to such contraction due to the lack of elasticity in capsular tissues. If the mistake is made of enclosing the fingers of an old person in plaster for a period of weeks, permanent stiffness of the fingers may result. Such trouble can be avoided merely by trimming the cast back to the proximal palmar crease in the hand, thus allowing; (1) a complete range of finger motion while the fracture heals; (2) good tendon motion; (3) adequate circulation to the extremity because the patient constantly pumps a sufficient blood flow to and from the fracture site.

When a fracture in the shaft of the ulna is suspected or present, care should be taken to include both the elbow and the wrist joints in the diagnostic x-ray to make sure that there is no luxation at either the superior or inferior radio-ulnar joint. Frequently, fractures in the shaft of the ulna are accompanied by dislocation of the head of the radius at the superior radio-ulnar joint or dislocation at the inferior radio-ulnar joint. If the fracture is treated without an x-ray of the elbow and wrist joints, a dislocation may be found months after the injury, at which time the doctor must resort to surgery and the chances of a good functional result are poor. If the dislocation can be recognized at the time of injury, it can usually be reduced easily and such reduction also helps to correct position of the fracture of the ulna.

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*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

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The reduction of dislocated epiphyses of the elbow in the growing child cannot be overemphasized. When the epiphysis of the capitulum is displaced, it is usually rotated nearly 180 degrees by pull of the extensors of wrist and fingers. If it is not replaced accurately, growth on the radial side of the humerus cannot keep pace with growth on the normal ulnar side. Therefore, months or years after the injury, cubitus valgus will be noted due to diminished growth on the radial side of the humerus. In addition to this deformity, the patient may complain of weakness in the upper extremity or may present the symptoms and signs of ulnar nerve palsy due to gradual stretching of the ulnar nerve around the increased angle of the medial side of the elbow joint. When the epiphysis of the medial epicondyle of the humerus is displaced, the flexors of the fingers and wrist and the pronator teres tend to hold the fragment distally and sensory ulnar nerve involvement is sometimes apparent within a few hours or days after the injury. If the epiphysis is not accurately replaced, involvement of the ulnar nerve by gradual pressure of the epiphysis or by inclusion in the organizing hematoma may cause a delayed ulnar palsy appearing months after the injury.

When the doctor is presented with a recent supracondylar fracture of the humerus with much swelling about the wrist, it is frequently a temptation to put the patient to bed and place the arm in traction in the hope that the swelling will subside and the elbow can be reduced with more safety later on. This is not a rational approach to the problem. The more swelling of the elbow, the greater is the indication for immediate reduction. It is only by an accurate reduction of the supracondylar fracture that pressure upon arteries, veins, lymphatics and nerves can be relieved. It is only by removal of such pressure that normal vascular channels can be reestablished and good pulse volume returned to the hand along with relief of lymphostasis and venostasis about the elbow.

Long oblique or spiral fractures of the lower third of the shaft of the tibia when treated conservatively tend to heal slowly or result in non-union due to poor blood supply. If union does take place, shortening of the tibia frequently follows, which results in pelvic tilt and often back complaints years later. Both tendency for delayed union and shortening can be corrected by the relatively simple technic of open reduction and insertion of one or two vitalium screws across the fracture site. By this procedure the fracture is anatomically reduced, healing is accelerated by the hairline reduction, and normal length of the extremity is established. Of course, a 48-hour preparation should be adhered to before operation and after operation a high-leg cast with the knee in 135 degrees extension should be applied for the usual period of three to four months or until adequate healing is present.

A SYMPOSIUM ON SODIUM AND WATER BALANCE*

DISTRIBUTION OF POTASSIUM AND SODIUM IN BODY

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The two important electrolytes of the body, from an osmotic standpoint, are potassium and sodium. They are present in solution in much higher concentration than other inorganic bases. For this reason they are also important in maintaining acid-base balance. In addition to these general effects they exert specific effects similarly to ions present in lower concentrations. They are so abundant in foods that deficiency of them is extremely difficult to produce in laboratory animals. They have, nevertheless, been shown to be essential.[1]

Changes in the amount or distribution of these ions produced by disease cause marked changes in the animal; for example, the high blood potassium in Addison's disease accounts for many of the symptoms[2] and the attacks of familial periodic paralysis, which are accompanied by low blood potassium, are relieved by administration of potassium salts.[3] In order to detect and evaluate such changes in the distribution of these ions, the normal distribution has been extensively studied. Such studies involve also the distribution of water, since the ions are present in solutions which are in osmotic balance. The information accumulated is still incomplete. The purpose of this paper is to present a scant summary of the available data on the distribution of these two elements.

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DISTRIBUTION IN CELLS

The potassium of the body is almost entirely intracellular. This was shown by Macallum[4] who precipitated the potassium in sections of tissue by means of sodium cobaltinitrite. Microscopic examination then revealed the location of the potassium precipitated *in situ*. His results may be summed up by saying: (1) the intercellular spaces are practically free of potassium, (2) the nuclei of cells are free of potassium, and (3) the distribution of the potassium in the cytoplasm is characteristic for each tissue. For certain tissues this distribution was as follows: in nerve fibers the potassium is in the myelin sheath, concentrated at the nodes of Ranvier and in Frohmans rings, the axon is free of potassium; in the acini of the pancreas the potassium is most concentrated on the side of the cells toward the lumen; in cartilage the potassium is scattered in the cytoplasm, there is none in the matrix; in smooth muscle the potassium is located at the periphery of the cells; in voluntary muscle the potassium is located in the anisotropic bands.

Fenn[5] has questioned the distribution in muscle cells shown by Macallum. He says that the potassium should be distributed like cell water and that it is not likely that a solution twice isotonic would be present in juxtaposition to distilled water in the muscle cell.

Scott[6] had confirmed the distribution shown by Macallum through the use of a micro incineration technic. The ash patterns found showed bands corresponding to the anisotropic bands of the muscle. This ash made at 650°C. should contain the potassium which boils at 760°C. Though Macallum's results might be explained as a Liesegang phenomenon[7] provided the precipitant could enter the fiber only at the end such penetration of the cell is improbable. There is no similar explanation for Scott's results. It seems unlikely that two such different technics should produce the same artefact.

The location of cell sodium has not been worked out though there is some evidence that most of it may be present in a bound state.

DISTRIBUTION BETWEEN CELLS AND EXTRACELLULAR FLUID

The only tissue which can be separated readily into extracellular and cellular parts for direct analysis is the blood. In human blood the sodium is present for all practical

TABLE 1

POTASSIUM AND SODIUM IN BLOOD

Animal	m.M./L.H_2O						
	Serum (92% H_2O)			Cells (65% H_2O)			
	K	Na	K+Na	K+Na	K	Na	K/Na
Dog (9)	5.5	150	155.5	149.9	8.9	141	0.063
Dog (10)	....	......	....	144.0	....	....	....
Cat (9)	6.0	156	162.0	148.5	8.5	140	0.060
Goat (9)	5.7	151	157.7	130.0	22.0	108	0.220
Sheep (9)	5.9	151	156.9	125.0	24.0	111	0.216
Cow (9)	5.9	152	157.9	124.7	23.7	111	0.213
Monkey (10)	....	......	....	159.0	....	....	....
Man (9)	6.0	163	169.0	165.5	131.0	34.5	3.800
Man (8)	5.4	158	163.4	166.?	166.0	?	....
Rabbit (9)	6.0	156	162.0	171.?	171.0	?	....
Pig (9)	6.2	149	155.2	163.?	163.0	?	....
Horse (9)	6.1	156	162.1	145.?	145.0	?	....

TABLE 2

POTASSIUM AND SODIUM IN ADULT ORGANS

Organ	H_2O %	m. eq./Kg. K	Na	K/Na	K+Na	m. eq./L.H_2O K+Na
Muscle (8)	79	93	31	3.00	124	157
Skeleton (8)	44	16	79	0.20	95	208
Skin (8)	73	27	70	0.38	97	133
Brain (8)	90	85	75	1.13	165	183
Liver (8)	79	55	82	0.67	137	173
Intestine (8)	85	70	......	......	......	......
Lungs (8)	78	39	109	0.36	148	189
Kidney (8)	80	45	72	0.62	117	146
Heart (8)	77	64	80	0.80	144	187
Heart (11)	79	67.5	61	1.11	128	162
Pancreas (8)	80	58	38	1.53	96	120
Uterus (8)	....	37	65	0.58	102	......

purposes exclusively in the plasma. If any at all is present in the cells it is a trace.[8] Earlier results shown in table 1 giving high cell Na[9] were probably due to contamination of the cells with plasma. In some other animals (notably the dog and cat) the sodium is in part in the cells. The erythrocyte sodium plus potassium in milliequivalents per liter of water is constant for all species, within the limits of error of the variations in analyst and methods, as one would expect, since the cell osmotic pressure is the same in mammals. The data shown in the table have been calculated, from the results given by the various authors, on the basis of milliequivalents per liter of water.

Even in the case of animals with a high erythrocyte sodium content, this element has been shown to be extracellular, with the exception of small amounts in muscle and larger amounts in the skeleton.[10]

TABLE 3

POTASSIUM AND SODIUM IN MUSCLE

Animal	H_2O %	m. eq./Kg. K	Na	K/Na	K+Na	m. eq./L.H_2O K+Na
Human, Adult (8)	79.0	93.0	31.0	3.00	124	157
Adult (11)	75.5	82.0	34.6	2.28	116	154
Monkey (10)	79.0	90.0	26.6	3.38	116	148
Pig (11)	72.9	65.0	68.0	0.95	133	176
Cow (11)	78.1	94.0	28.4	3.30	122	155
Steer (11)	79.8	68.5	45.5	1.50	134	168
Calf (11)	75.3	97.7	36.0	2.68	134	178
Deer (11)	75.2	86.0	30.6	2.70	117	156
Rabbit, thigh (11)	76.8	102.0	19.9	5.10	122	163
red muscle (11)	74.9	109.0	25.6	4.25	135	180
white muscle (11)	74.9	97.5	19.1	5.10	117	157
Rabbit (10)	80.0	78.0	29.0	2.68	101	127
Dog (10)	68.0	67.4	30.2	2.24	98	144
Dog I, thigh + back (11)	76.4	83.5	41.0	2.02	124	163
Dog II (11)	74.8	83.5	21.4	3.90	105	141
Cat, thigh + back (11)	75.1	98.0	31.8	3.08	130	173
Hen, breast + thigh (11)	69.3	119.0	41.5	2.86	160	203
Cock, red muscle (11)	75.4	95.5	33.5	2.86	129	171
white muscle (11)	73.1	105.0	33.8	3.11	139	190
Turkey, red muscle (11)	73.8	93.0	25.2	3.78	118	163
white muscle (11)	72.5	94.0	18.5	5.09	112	155
Frog, R. esculenta (11)	81.6	79.0	24.0	3.28	103	126
R. Catesbiana (11)	79.8	89.5	23.3	3.86	113	142

This tissue distribution has been determined indirectly. Chloride which is extracellular since cells are, for the most part, anion impermeable is used to calculate the amount of extracellular water in the tissue. Total tissue chloride/plasma cl. concentration=extracellular water. In tissues, with the exception of muscle and skeleton, the sodium present is accounted for by the extracellular water on the assumption that it has a sodium concentration like that of plasma. The excess sodium, above that accounted for by the "chloride space," in bone is not extractable with water or strong potassium hydroxide so is apparently bound in some way.

POTASSIUM AND SODIUM IN VARIOUS ORGANS

Since the potassium is intracellular and the sodium is extracellular and the two solutions are in osmotic equilibrium the K m. eq. per kg/Na m. eq. per kg. is the ratio intracellular water/ extracellular water. The K m. eq. per kg. + Na m. eq. per kg. is a measure of the total water content and K m. eq. per L. H_2O + Na m. eq. per L. H_2O is an indication of the part of the osmotic pressure accounted for by these two ions. Table 2 shows these relations for various adult human organs. The K/Na ratio is highest in the case of muscle and excluding the skeleton is lowest in the case of skin and lungs. The muscle then has about three times as much cell water as extracellular water while the skin and lungs have a reverse situation.

The electrolyte concentration of muscle has probably been studied more than that of any other tissue. Table 3 shows comparative data calculated from the literature. The variations shown here in K+Na are no greater than the variations reported for homologous muscles or even different parts of the same muscle. In general the K/Na ratio is high. The values given in the tables must be considered as average values rather than as fixed normals. There is considerable change in muscle electrolyte on contraction, with loss of potassium and gain of sodium by the cells. Loss of potassium during activity has been reported also to occur from heart muscle, nerve fibers and ganglia. Such changes probably occur in other organs during active functioning also. The one exception, which has been demonstrated, is the erythrocyte. Potassium cannot enter or leave the erythrocyte unless the cell is injured[12,13]. This injury is least in defibrinated blood, less in oxalated or citrated blood than in inorganic solutions and in inorganic solutions is roughly in proportion to their departure from average blood composition. The erythrocyte is anomalous also in that it is permeable to chloride.

POTASSIUM AND SODIUM OF WHOLE ANIMAL BODIES

Table 4 gives the potassium and sodium relations of whole bodies of various species.

TABLE 4

TOTAL BODY POTASSIUM AND SODIUM

Animal	H_2O	m. eq./Kg.				m. eq./L.H_2O
	%	K	Na	K/Na	K+Na	K+Na
Human, Fetus 5 mo. (8)	91	51	112	0.45	163	179
Fetus 6 mo. (8)	87	41	94	0.43	135	155
Fetus 7 mo. (8)	86	48	93	0.51	142	165
Premature 7 mo. (8)	85	44	105	0.42	149	176
Newborn (8)	80	49	78	0.63	127	159
Adult (8)	72	68	48	1.42	116	161
Monkey (10)	68	64	55	1.16	119	174
Dog (10)	59	48	55	0.88	103	175
Dog (14)	....	....	......	1.468	......	......
Cat 29 days (14)	....	....	......	1.297	......	......
Rabbit (10)	74	63	48	1.31	111	148
Rabbit 14 days (14)	....	....	......	0.835	......	......
Embryo (14)	....	....	......	1.272	......	......
Mouse (14)	....	....	......	0.787	......	......

The figures for the human show that the total water in the fetus is much higher (K plus Na per kg.) than in the adult and that much more of the body water of the fetus is extracellular (K/Na). In most adult animals the ratios are similar to those of the adult human. When the whole body is considered the excess sodium in the skeleton and muscle above that accounted for by the chloride space will introduce some error in the correspondence of the K/Na m.eq. per kg. ratio to intracellular water/extracellular water ratio.

The data from which these tables were calculated are in many instances inadequately stated for evaluation of their validity as standards. In many instances the number of animals was not stated, in some of these the analyses were apparently done on a single animal or single samples, so that in no case can these figures be accepted as absolute values but as averages or less.

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BOUND WATER

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The subject of bound water concerns itself with something about which there is so much doubt that it appears difficult even to attempt a definition. Bound water may be considered to be a synonym for water of hydration but it bears special reference to colloids, especially proteins; thus it concerns itself with water molecules which are combined with dissolved protein in such a way that the water becomes a part of the molecule kinetic unit of the protein. Under such circumstances this water is said not to retain its normal solvent properties nor does it participate to any extent in osmotic transfers so that it presumably possesses different physical properties than the surrounding free water.

It would seem necessary at least to note the complexity of structure of even free water. The molecular structure of liquid water is assumed to consist of hydrogen and oxygen atoms joined in the form of a "V" with oxygen at the juncture and H at each end. Further, these molecules are polymerized in varying numbers, either as dimers, trimers, or even higher polymers, the number of units depending upon such factors as the temperature and the types and concentrations of varius ions which may be present. X-ray diffraction studies show that, statistically the most common polymeric structure of liquid water is a tetrahedron consisting of four water molecules. At this point a quotation from an old chemistry textbook would seem appropriate: "Water is water, not a single substance in the proper acceptation of the term, but a mush or a mixture; an entirely peculiar substance, not to be represented by any single formula nor spoken of by any single name."

Many types of hydration are possible, ranging from simple mechanical entanglement to a closely packed dielectrically saturated water in an intense dielectric field. The degree and extent of controlling factors determining the type of hydration is imperfectly known. One well known example of hydration is the chemical combination of H_2O with NH_3 wherein a hydrogen bond is established between the N of the NH_3 and the O of the H_2O. But water combinations other than chemical bond formations involving the attraction of H_2O with other molecules are not well understood.

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Necessary evidence for hydration in crystals is the presence of stochiometrical proportions of H_2O. A solid crystalline hydrate may not exist in solution, however, because solution breaks up the crystal structure. Furthermore, water does not take part in the crystalline structure apparently because during dehydration of a crystal, the structure is retained in the face of parallel increases in density and decreases in volume.

Possible means of hydration of a protein involve water combination by the formation of covalent bonds between water and the OH, COOH, NH_2, and NH groups of protein sidechains. These hydrophilic sidechains frequently serve as connecting links between polypeptide chains composing the protein molecule. Factors determining the extent of hydration of a protein molecule include the ratio of hydrophilic to hydrophobic sidechains, differences in ionic size, (especially great in the case of protein and water, thus tending to favor hydration) acidic or basic characteristics of sidechains which determine their efficacy as coordination centers for orientation of water dipoles, and finally, the isoelectric point where zwitterionic action may serve to approximate active protein groups thus preventing hydration by forcing out the solvating water. This latter point is illustrated in the case of gelatin at the isoelectric point where water imbibed during swelling retains its solvent properties and so is not hydrated or bound.

The binding of water by a protein is therefore easily visualized but is not so easy of proof. Several methods have been suggested for proving or disproving the presence of bound water, many of which depend upon the measurement of the colligative properties of bound and free water. The thermodynamic data thus obtained are reliable but their interpretation is open to doubt because of many complicating factors, not the least of which is the relation of water molecules to each other. The first of these methods involves adding a reference substance, such as sucrose or urea, to a gelatin solution and to a volume of water equal to that in the gelatin solution and then comparing the decrease in freezing points or in vapor pressures which should occur. If any water were bound by the gelatin, one should expect a greater decrease of vapor pressure or freezing point, but in actuality, no such difference has ever been observed since the freezing points and vapor pressures are lowered by the same amount in both solutions. This has also been found to be true for muscle tissue, blood, and egg white. A second method involves ultrafiltration wherein a reference substance such as glucose is added to a gelatin solution and this solution is then subjected to ultrafiltration, which should remove all water. This reference substance would be assumed to diffuse freely throughout the free water but not in the bound water. This experiment denies the presence of bound water, for the concentration of glucose in the ultrafiltrate is exactly the same as that in the original solution. A third method utilizes the separation of ice crystals following cooling of water. One would expect bound water not to freeze. It has been found with gelatin gel that a solid solution separates out at about -20° C. and the composition remains constant down to at least -45° C, with about 0.5 grams H_2O per gram of gelatin contained thus arguing for bound water. However, even as one of the most avid proponents of bound water admits, it takes a long time for equilibrium to be established between ice crystals and water—as long as 18 days —and these conditions are rarely observed. More importantly it is very probable that all of the water did not freeze out of the gelatin because it was supercooled and supercooled water will not ordinarily freeze unless first seeded by ice crystals which, in this case, could not penetrate from the outside of the gelatin mass. A fourth method takes advantages of the property of birefringence which measures the regularity of positioning of protein chains and by its diminution, in the case of hydration, indicates disorientation of the molecular structure due to the presence of water molecules. Finally, x-ray diffraction pictures of hydrated proteins show an increased intramolecular spacing following hydration indicating the presence of water combined via the hydrogen bonds to the extent of about 0.4 gram H_2O per gram of protein. However these last two methods tell us only of the presence of water and not necessarily of the mode of combination or the nature of the combined water.

Accepted values (by those who believe in it) of protein-bound water vary from 0.2 gram H_2O to 0.5 gram H_2O per gram of protein but reported values range from 0.08 gram H_2O to 7 grams H_2O per gram of gelatin. The extent of hydration of proteins must therefore remain undecided and since the present trend of evidence indicates that there is little if any hydration of proteins for practical purposes all the water in protein solutions can be considered to be solvent water. There has been, as yet, no method developed to give precise information on the presence of bound water and we cannot be certain that water can form an integral part of the molecular-kinetic unit of intracellular colloids.

Certain practical aspects of bound water may be mentioned at this point, that is if there is any such thing as bound water. From x-ray data it appears quite certain that water is combined with protein and lipoid but whether this water is free or bound is uncertain. Let us then refer to this water as combined water which is probably not a better term but at least does not commit us to think of it as inactive water. One feature of such combined water occurs in the salting out effect of certain ions. These ions precipitate proteins by virtue of the fact that their affinity for water is greater than protein, and, by their presence, they dehydrate protein. Na and SO_4 are the physiologically important ions with the greatest affinity for water and Na_2SO_4 is one of the best protein precipitants. Another important role of combined water is in membrane structure. X-ray studies of myelinated nerves and erythrocytes show that these structures consist of bimolecular layers of lipoid which extend outward into a water layer on each side which separates one set of lipoid layers from its neighbor. When basic histone protein molecules are present, they may cause complete expulsion of the water layers by replacing them since they easily react with polar acid groups of the lipoid cephalin. Myelinated structures have been shown by histologists to possess channels communicating with the axis cylinder and although these might be fixative artefacts, they may also be correlated with water layers shown to exist by x-ray studies in brain lipoid mixtures. These spaces are found to vary according to the per cent of water in the solution. X-ray studies, interestingly enough, show the same degree and type of water layer disposition between molecules of cephalin-histone, thromboplastin, and blood platelets, indicating the structural relationship between these substances so important and similar in their actions in blood clotting. Finally, most germane to the general subject of the symposium, it has been shown, by x-ray studies, that the mitochondria of cells of the distal tubule of the amphibian kidney show a spacing between their lipoid-protein components of 42° Angstrom units in diffraction pictures. This relatively large spacing indicates a high degree of solvation of the polar groups of the lipoid and protein and may be important in the role of these cells in water transport from tubule lumen to tubule blood.

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SODIUM AND POTASSIUM IN RELATION TO THE ACTION OF DIURETICS

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A complete discussion of the relation of sodium and potassium to the action of diuretics is impossible in the time available. Consequently the subject will be presented in the form of illustrations of the importance of the ions of these elements in diuretic action. Citations from the literature are chosen arbitrarily without attempting to make an extensive survey of pertinent publications.

SODIUM AND POTASSIUM SALTS AS DIURETICS

It is well established that there is a distinct difference between the behavior of sodium and of potassium salts with respect to their action as diuretics. Potassium has a much lower renal threshold than sodium. In other words after filtration through the glomerulus reabsorption by the tubules is much greater in the case of sodium than of potassium. Potassium, being retained in the filtrate, retains water with it by osmotic action resulting in diuresis. The rapid elimination of potassium is also evidenced by the rapid lowering of plasma potassium concentrations after administration of its salts or by the failure of blood concentrations to show any appreciable rise after administration of potassium salts by mouth.

This may be illustrated from an experiment of Keith and Binger.[1] Two normal subjects were placed on a fixed diet low in sodium chloride with extra fluid restricted to 800 c.c. daily for six days. Urine volume, urinary sodium, potassium and chloride, and serum potassium were determined. One subject was then given sodium chloride and one potassium chloride daily for five days in doses approximately equivalent from the molar standpoint (a total of 0.6 mols in five days). A post experimental period was also followed. A distinct difference was evident in the results of the two subjects. With sodium chloride the urine volume rose by from 100 to 200 c.c. daily but only on the last two days of the five day period. With potassium chloride the urine volume rose by slightly more than 300 c.c. daily, the rise beginning on the second day. There was a still greater difference with respect to urine chloride. On sodium chloride administration the urine chloride rose gradually throughout the five day period from a normal daily output of 20 to a maximum of approximately 180 milliequivalents. In contrast, on potassium chloride administration the chloride excretion rose from a similar control value to a prompt and sustained daily output of approximately 150 milliequivalents. As to be expected, sodium excretion paralleled chloride excretion with sodium chloride and potassium excretion paralleled chloride excretion with potassium chloride. There was also a slight increase in sodium excretion with the potassium chloride. In other words the total excretion of both water and salt was definitely greater with potassium chloride than with sodium chloride administration during the experimental period. In another series of cases in which potassium chloride was administered the urinary potassium showed concentrations up to 50 times those in the plasma. Values for plasma concentrations, on the other hand, showed little change varying from 30 to 40 mg. per cent.

As a matter of fact the slow elimination of sodium may actually promote the development of edema rather than diuresis since sodium chloride if not excreted will hold a proportionate amount of extracellular water in order to maintain isotonicity of extracellular fluids. Torbert and Cheney[2] produced puffiness of the eyelids and edema of the ankles in two normal subjects by administration of 50 mgs. of sodium chloride daily, for two days. This was associated with a decrease in plasma protein concentration, in hematocrit and in colloidal osmotic pressure of the serum the latter finding supposedly contributing to the causation of the edema. These findings are, of course, in keeping with the retention of extracellular water. It is also a common experience that in patients with edema the withholding of sodium chloride will result in diuresis. Stockton[3] placed two edematous patients on a salt free diet without other changes in the treatment. During the first ten days the urine volume increased 77.2 per cent above that of a previous control period on an ordinary diet. During the second ten day period the increase was 56 per cent over the volume during the control period. The blood chloride and daily chloride excretion showed little change in spite of the salt free diet. With the increased urinary volume, its chloride concentration was proportionately decreased.

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In addition to the behavior of sodium and potassium salts per se they apparently play some part in the action of certain other classes of diuretics.

ACID PRODUCING SALTS SUCH AS AMMONIUM CHLORIDE AND CALCIUM CHLORIDE

These acid producing salts supply chloride to the body in excess of base since calcium is precipitated in large part in the intestine as bicarbonate and ammonia is changed to urea after absorption. This results in effect to an increase of basic ions in the body in the form of chloride and a decrease as bicarbonate producing a relative acidity. The concentration of base in the plasma remains almost stationary although it is somewhat lowered in the absence of edema due to increased excretion.

Gamble, Blackfan, and Hamilton[4] investigated the sodium and potassium exchanges under the action of acid producing salts. To illustrate this one experiment may be cited as follows: three children with edema and one without edema were placed on a fixed diet low in sodium chloride. After a control period without medication they were given calcium chloride over a three day (patients with edema) or five day (no edema) period. The average daily excretion of base for four days following the medication in excess of the excretion during a control period was as follows:

Expressed as c.c. of 0.1 N Solution

	Total Fixed Base	Na	K	Ca	Mg.
No edema	1242	455	569	284	37
Edema	2200	1238	751	157	54

The relatively greater increase in excretion of base in edema with no change in plasma concentration might be expected, since the edema fluid which is mobilized is a source of base. It is assumed that the potassium excretion represents elimination of intracellular water. This is in keeping with the relatively high potassium excretion in the absence of edema fluid where excess sodium and extracellular water would not be available.

This excess excretion of sodium and potassium under the action of acid producing diuretics is undoubtedly a factor in the elimination of water by virtue of their osmotic effects in the renal tubule. Of course the explanation of the freeing of the base by increased acidity is another problem.

RELATION OF SODIUM AND POTASSIUM TO THE ACTION OF MERCURIAL DIURETICS

It is generally accepted today that the important action of mercurial diuretics is by interference with renal tubular absorption[5, 6]. In the literature, however, there is much evidence that a general tissue effect plays some part in this diuretic action and some investigators have even believed this to be the main action. The general tissue effect of the mercurials involves a freeing of potassium or sodium chloride from the tissues into the plasma, and the excretion of this excess salt plays a part in the elimination of water. This may be illustrated by the work of Stockton.[3] Six edematous patients were maintained on a constant low salt diet (2 gms. of sodium chloride and 1200 c.c. of fluid) until blood and urine findings were constant for three days. Then they were given merbaphen (one dose) and the changes were as follows: Diuresis (+300 per cent) began in two hours with peaks at five and 13 hours after administration. Some diuresis continued up to 30 hours. Blood chloride increased within two hours with a peak of +6 per cent in five hours but fell below the control level in 11 hours after administration. A second peak occurred in from 18 to 21 hours (up to +4 per cent). The excretion of chloride paralleled the diuresis, the rate of excretion going as high as +2800 per cent over the control period. What is more remarkable is that the urinary concentration of sodium chloride increased in spite of the greater volume of urine, being 40 per cent above the control value during the whole period of diuresis and going as high as +75 per cent. Similar results were obtained with salyrgan on five patients but the changes involving all factors were even greater than with merbaphen.

The changes in blood chloride were slight however and Keith and Whelan[7] failed to find any constant change under the action of mercurial diuretics. If such does occur it would indicate an extrarenal action of mercurial diuretics. It is also common experience that mercurials may fail in the presence of low plasma chlorides and that the diuresis may be increased by the administration of acid producing chlorides.[7] Whether this increase could be produced by raising the blood chlorides by the administration of sodium chloride as such, without the acidity factor is doubtful. I am aware of no evidence that such is the case. Irrespective of this factor the markedly increased excretion of chloride must contribute to the increased urinary volume in the form of saline diuresis.

RELATION OF SODIUM AND POTASSIUM TO THE ACTION OF XANTHINE DIURETICS

The xanthine diuretics like the mercurials are believed to act essentially on the kidney by increasing filtration through circulatory changes or by interferences with tubular reabsorption. However here again, the diuresis is associated with sodium chloride exchanges which may well have some role in the action and which have been assigned predominant importance by some investigators. Stockton[3] also used theophylline on six edematous patients producing diuresis on the average from the 4th to 35th hour after administration. Blood chloride changes were irregular being both above and below normal during this period. The rate of chloride excretion however paralleled the diuresis, reaching a peak of 600 per cent above that of the control period and again in spite of the diuresis the chloride concentration in the urine was also above that in the control period going as high as 40 per cent above normal. Curtis[8] demonstrated that he could prevent the diuretic action of theophylline ethylenediamine in rabbits by injecting distilled water into the peritoneal cavity. The response to the diuretic was restored only after the peritoneal fluid had become isotonic. During this period of inhibition the small amounts of urine were low in chloride but when diuresis was resumed (presumably after sodium chloride had ceased going into the peritoneal fluid) the urine was high in chloride both in concentration and total amount. In these experiments also the diuresis was accompanied by raised blood chlorides. Consequently a mobilization of sodium or potassium chloride into the plasma from the tissues may be a factor in the diuretic action of the xanthines, and as with the mercurial diuretics the increased chloride excretion would tend to increase urine volume.

SUMMARY

The salts of potassium are definitely more diuretic than sodium salts. The latter, under certain conditions may actually promote the retention of edema fluid. There is evidence that an increased excretion of sodium and potassium chloride, although possibly not of major importance, is a factor in the diuretic action of the acid producing salts, the mercurials, and the xanthines.

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THE ROLE OF THE ADRENAL GLAND IN SODIUM AND WATER EXCHANGE

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Since our interest at this time is in the control of salt and water exchange, it is fitting to discuss the adrenal gland. The adrenal cortex has a profound influence upon the salt and water exchange of mammals. This is only one phase of the multiple functions of the adrenal cortex. As is well known, the adrenal glands are essential for life. They are necessary for normal protein and carbohydrate metabolism as well as for the maintenance of the general body resistance to stress.

When operative technics for adrenalectomy were perfected sufficiently so that the animals died of adrenal insufficiency several days after the operation rather than immediately from operative shock, it was noted that as adrenal insufficiency developed there was a fall in the serum sodium and chloride.[1] We are omitting such animals as the opossum and the elasmobranch fishes from our discussion since they do not show the "typical" alterations in sodium exchange which are under discussion here. Attempts at the utilization of these facts bore excellent fruit and it was found that proper therapy with sodium salts could maintain adrenalectomized animals in good condition.[2,3]

These studies were extended to patients with Addison's disease and it was found that they too could be improved by the administration of sodium salts.[4,5] The converse also was found to be true and salt restriction was applied as a diagnostic test for patients with a question of Addison's disease.[6]

The defect in the kidney appears to be an impaired ability to resorb sodium ion from the glomerular filtrate. An intriguing alternate theory has been advanced, namely, that the kidney has an impaired ability to produce ammonia. This concept is supported by the fact that when sodium excretion declines under proper therapy, the urinary ammonia content increases.

The renal wastage of sodium ion is accompanied by diuresis, which decreases as adrenal insufficiency advances and terminates with oliguria or anuria.[7] Despite the excessive urinary loss of water, the adrenalectomized animal and the patient with Addison's disease do not respond to the administration of water with diuresis. The mechanism of this loss of the diuretic response to water is not entirely clear. The adrenalectomized rat shows a delay in the time required for the stomach to empty and a deficit in intestinal absorption, but this is not the entire picture since injected water also fails to produce diuresis.[8] In addition, symptoms of water intoxication appear in the adrenalectomized animal at lower levels of water retention than in normal animals.[9]

This failure to produce diuresis is the basis for another test for adrenal hypofunction.[10] This test has a high degree of reliability and there is little apparent danger. In this procedure a measured amount of water is administered to patients suspected of having Addison's disease. If the disease is present no diuresis occurs; if not, there is a normal diuretic response to the administered water.

These urinary losses of sodium and water are reflected in a contracted circulating blood volume which is poor in sodium. Sodium ion and water then migrate from the interstitial fluid reservoirs; this creates a differential in the osmotic pressure between the body cells and their fluid environment which is naturally immediately compensated for by migration of water into the cells.[11]

As might be anticipated, these defects in sodium and water balance can be corrected by appropriate therapy with adrenal cortical extracts. Desoxycorticosterone and the amorphous fraction are particularly potent in this regard.

It has been reported that aqueous adrenal extracts can be fractionated into two fractions; one is capable of maintaining life and the other affects sodium metabolism. It has further been demonstrated that it is possible to keep animals alive and in apparently excellent condition except for the low serum sodium and excessive urinary sodium loss characteristic of a severe state of adrenal insufficiency.

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Of the cortical steroids, desoxycorticosterone has the most pronounced effect on salt and water metabolism. Indeed, it is so potent that it readily produces extreme sodium retention and loss of potassium and water to such a degree that muscular weakness and paralysis and even cardiac failure may occur when it is administered too vigorously.[12] When large doses of desoxycorticosterone acetate are given for a long period of time, a diabetes insipidus-like syndrome develops.[13] This is, of course, very different from diabetes insipidus since it is unaffected by pituitary antidiuretic hormone and restriction in the fluid intake does not produce dehydration. The feeding of a salt poor diet reduces the severity of the polydipsia and the polyuria. The explanation for this entire phenomenon may be that the extracellular sodium concentration is greatly increased and cellular hydration is, therefore, decreased. This is diametrically opposed to the case in adrenal insufficiency. The water intake is then greater because of an increased thirst resulting from the effects of the intracellular dehydration. In other words, the polyuria may merely be a reflection of an induced polydipsia.

The corticosterones without a hydroxyl group in the 17 position also cause sodium retention but to a lesser degree than does desoxycorticosterone. However, the introduction of a 17 hydroxyl group, as in Kendall's compound E, enhances the carbohydrate effect but reverses the effect on sodium and potassium; that is to say, the administration of compound E to an intact animal causes the renal loss of sodium and water similar to adrenalectomy and opposite to the effect of the other adrenal steroids.[14]

In the adrenalectomized animal there is also a decrease in the intestinal absorption of sodium so that there is more than just a loss of sodium ion contributing to the low serum sodium.[15]

SUMMARY

The adrenalectomized animal shows a marked loss of sodium and water in the urine which is reflected in a decreased circulating blood volume and a low serum sodium content. This, in turn, leads to a decrease in the sodium and water concentration in the interstitial fluid compartment. This is further exaggerated by the shift of water into the cells which had a higher osmotic pressure than their bathing fluids. These effects are somewhat exaggerated by an inability to absorb sodium properly from the intestinal lumen.

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THE ANTIDIURETIC HORMONE*

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All organisms which live on dry land or in hypertonic salt water possess mechanisms which enable them to conserve body water. Birds and mammals have met the problem by the development of a device which insures the reabsorption of practically all the water and much of the electrolyte content of glomerular filtrate. This device is a thin segment of tubule (Henle's loop) which lies between two convoluted portions of the kidney tubule; here, the major part of the glomerular filtrate is reabsorbed into the blood stream with the assistance of an antidiuretic hormone (ADH) secreted by the neurohypophysis. Urine volume is, therefore, regulated in large part by the action of an endocrine product upon a specific portion of the renal tubule.

Diabetes insipidus represents the loss of a biologic adaptation occasioned by failure of the neurohypophysis. Other things being equal, the rate of urine flow appears to be inversely proportional to the concentration of antidiuretic hormone circulating in the blood, and complete destruction of the neurohypophysis results in a maximal diuresis of 20 to 25 c. c. per minute for an average adult. Since the average rate of glomerular filtration is about 125 c. c. per minute,[1] it is obvious that the reabsorption of approximately 80 per cent of the glomerular filtrate is not under neurohypophyseal control. Even excessive amounts of ADH, however, will not produce complete anuria, for pitressin facilitates the reabsorption of only that water in excess of the amount required to keep the urinary solids in solution.[2] For this reason, pitressin is ineffective in controlling the polyuria in chronic nephritis, diabetes mellitus or salt diuresis.

The chemical nature of ADH is not well understood[3] and there is no general agreement as to the number of separate hormones manufactured by the neurohypophysis. It must be remembered that extracts of other organs possess antidiuretic properties—liver[4], kidney[5], and placenta.[6]

It has been clearly demonstrated that the secretion of ADH is controlled by nonmyelinated nerve fiber arising chiefly from the paired supra-optic nuclei but probably also from the paraventricular nuclei; these fibers supply the pars nervosa and with it form a functional unit. The hormone is probably manufactured by special cells in the median eminence, stalk and posterior lobe called pituicytes.[7] It has also been extracted from the supra-optic region of the dog's hypothalamus.[8] The hypothalamic nuclei are, of course, intimately connected with various parts of the central nervous system so that there are innumerable routes for impulses affecting postpituitary activity. For example, Chang and his associates[9,10] have reported that stimulation of the central end of the cut vagus results in disappearance of the secretory granules in the pituicytes and increased secretion of ADH. Boyd, Lee and Stevens[11] have claimed to produce antidiuresis by stimulating the retina of rats with flashes of light. Other examples of the reflex liberation of pituitrin have been recorded by Rydin[12] and Haterius.[13] The effects of exercise, emotion, sensory stimuli, hypnosis, conditioned reflexes and central nervous system depressants upon water excretion have been reviewed by Pickford.[14] Acetylcholine,[15] morphine[16] and nicotine[17] have been shown to exert an antidiuretic action through the mediation of the neurohypophysis and it may be that the xanthines have an opposite action. Destructive lesions of the supra-optic nuclei or of the hypothalamico-hypophyseal tracts result in atrophy of the pars nervosa, in a great diminution in the amount of antidiuretic material which can be extracted from the posterior lobe, and in polyuria.[18] Destruction must be nearly complete, however, if appreciable polyuria is to result; about 85 per cent of the cells of the supra-optic nuclei must be destroyed before diuresis appears in the rat,[19] the monkey[20, 21] and the dog.[22] From experiments on dogs with diabetes insipidus showing that dehydration does not result in shrinkage of the volume of extracellular fluid, Shannon[23] concluded that the absence of ADH results in more complete tubular reabsorption of sodium as well as diminished reabsorption of water. Although it seems that maximal polyuria can be produced in experimental animals by complete destruction of the neurohypophysis even in the absence of the anterior lobe, it also seems unlikely that such complete destruction often occurs in human diabetes insipidus and that, therefore, the diuretic property of anterior lobe tissue is of clinical importance.

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It may be supposed that the secretion of ADH will depend upon the need for water conservation, and a considerable amount of experimental work (for bibliography see Pickford[14]) has been done which seems to prove that dehydration results in histologic changes in the neurohypophysis, in diminution of the amount of ADH extractable from the pars nervosa, and in the appearance of ADH in the urine. There are grounds for suspecting, however, that the antidiuretic material which appears in the urine may not actually be pitressin.[6, 24, 25]

The train of events which follows the administration of large amounts of water is perhaps better understood. Rioch[26, 27] was the first to note that a small but measurable hemodilution occurs after water ingestion and that there is a time-lag of about twenty minutes between the peak of blood dilution and the peak of maximum diuresis. This phenomenon has been confirmed repeatedly and it also occurs after the intravenous administration of water.[28] It is fairly well agreed that the stimulus to water excretion consists in a relatively abrupt change in the osmotic pressure of plasma[29] but the precise mechanism whereby blood dilution increases urine formation is still open to debate. Klisiecki and associates[30] formulated what might be called the modern theory of water diuresis by postulating that hemodilution inhibits the production of ADH and that the latent period is due to the time required for the concentration of the hormone in the blood to fall below a critical level. A large body of indirect evidence is interpretable from this viewpoint, and Hart and Verney[31] have even calculated that diuresis in man will result from a fall in the plasma concentration of ADH of less than one part in fifteen billion. On the other hand, it has been shown that administration of hypertonic saline solution stimulates the production of ADH.[32, 33] Hare and co-workers[34] confirmed these findings and, by relating the concentration of chloride in the water reabsorbed by the renal tubules to the concentration of chloride in the plasma, found that pitressin does not affect chloride excretion. Chambers[35] reported that the administration of salt produced edema and a diminished hormone content of the neurohypophysis. Hickey and Hare[36] extended these observations to man and demonstrated that the intravenous injection of hypertonic saline solution increases ADH secretion. They feel that the inhibition of a pre-existing water diuresis by salt injection is good evidence that the neurohypophysis is functioning properly. As a means of distinguishing polyuria due to true diabetes insipidus from that secondary to polydipsia, they propose a test which consists in infusing 2.5 per cent sodium chloride at a standard rate during a well established diuresis. With the aid of simultaneous plasma inulin clearance determinations they calculate the concentration of chloride in the water reabsorbed by the renal tubules and compare it with the concentration of chloride in the plasma (R/P). Pituitrin (endogenous or exogenous) causes the tubules to reabsorb water in preference to chloride and diminishes the ratio to less than 1.0; in the absence of functioning pitressin-forming tissue no antidiuresis occurs and the R/P ratio does not diminish. Shannon[37] has estimated that a dog weighing 10 to 15 kg. produces ADH at the rate of 0.001 to 0.005 units per hour.

Attractive as this theory is, the evidence is not entirely in its favor. It has been shown, for example, that urine flow may remain at low levels in decerebrate animals from which all pitressin-forming tissue has been excluded and that in these preparations the events leading up to water diuresis occurs in a perfectly normal manner.[38, 39, 40] White and Findley[41] found that a similar lag occurs in the renal excretion of chloride and glucose but not of creatinine; they felt that the latent period for all these threshold substances might be due to the time required for the renal parenchyma to become adjusted to new osmolar concentrations and that there is no need to resort to the hypopitressinemia theory. Obviously, more work is needed on this problem.

It should be stated that no clinical syndrome has been described which may reasonably be attributed to chronic hyperfunction of the neurohypophysis.

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THE DIURETIC HORMONE

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The title of this paper is provocative rather than descriptive. Is there a diuretic hormone? Is there a hormone which is the antagonist of the antidiuretic hormone of the neurohypophysis? Are there possibly a number of diuretic hormones? Or, perhaps, the term is a misnomer and it would be better to speak of the "diuretic forces" to represent the non-specific effect of the various catabolic agents producing waste products which tend to augment urine flow.

This paper represents no new material. Certain concepts will be reviewed and evaluated.

The disturbed physiology of diabetes insipidus has been discussed in a previous paper in this symposium. It was to be expected that a diuretic hormone would be sought for as an explanation for the profound diuresis seen in diabetes insipidus. It became apparent, however, that the diuresis of diabetes insipidus was caused by a deficiency of the antidiuretic hormone of the neurohypophysis.

In 1918 von Hann[1] pointed out that in man the pars anterior of the hypophysis must be at least partially intact if destruction of the neurohypophysis is to result in diabetes insipidus. It is of interest to examine the evidence for this concept. Von Hann reviewed the necropsy findings in 20 cases of diabetes insipidus. The pars anterior was either wholly or partially intact while the neurohypophysis was "destroyed" in 17 of these cases. In the three remaining cases the state of the pars anterior was not noted. This group of cases was contrasted with nine cases of Simmond's disease in which there was said to be a total destruction of the hypophysis; diabetes insipidus was not encountered in any of these cases.

The concept of von Hann has had wide clinical support through the years. Diabetes insipidus and Simmond's disease have appeared to be mutually exclusive. Any number of clinical cases[2] have been described in which diabetes insipidus was succeeded by Simmond's disease with amelioration of the diabetes insipidus. At necropsy total destruction of the hypophysis is described. It has been assumed that the clinical sequence was caused by a progressive lesion involving the hypophysis which destroyed first the neurohypophysis producing diabetes insipidus and then spread to the pars anterior causing Simmond's disease and "curing" the diabetes insipidus.

There has recently been reported a case of severe permanent diabetes insipidus occurring in what was said to be pituitary dwarfism.[3] At necropsy bilateral localized lesions in the hypothalamus with complete destruction of the neurohypophysis were described. The hypothalamic lesions were limited almost entirely to the region of the supraoptic nuclei. There was almost complete disappearance of the cells of the paraventricular nuclei. Careful histologic study of the pars anterior revealed fibrosis; the total number of cells was estimated at about one-sixth of normal.

It would appear then that if the von Hann concept is correct, a relatively small fraction of the pars anterior may suffice for the production of diabetes insipidus provided there is adequate destruction of the neurohypophysis.

Experimental support for the von Hann concept was slow to appear. In 1934 Richter[4] described his work in rats. He produced diabetes insipidus by lesions of the supra-optico-hypophyseal system. He was unable to produce permanent diabetes insipidus unless some functioning pars anterior remained intact in the animals. These experiments were confirmed by others[5]. However, in 1941, Heinbecker and White[6] were able to produce permanent diabetes insipidus in nine dogs by total destruction of the neurohypophysis and concomitant removal of the entire pars anterior. They confirmed the fact that there was no pars anterior intact at all by careful histologic studies. They made meticulous studies of the number of intact cells in the supra-optic nuclei in their experimental animals and they concluded that the survival of less than 3 per cent of intact cells in the supra-optic nuclei was the key feature in the production of permanent diabetes insipidus in dogs.

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These rather conclusive experiments bring up a number of important points. Heinbecker and White are very critical of previous work because of inadequate histologic evaluation of both experimental and clinical lesions. Their criticisms are just. However, without further studies it is not justifiable to carry over the results in the dogs to the rats and man. Our comments will then be limited to the dog; it should be noted that the experiments of Heinbecker and White do not rule out a possible diuretic hormone produced by the pars anterior nor do they eliminate the possibility that the pars anterior may act as a diuretic force. The fact is that the pars anterior is not essential for total diabetes insipidus in the dog if there is "total" destruction of the neurohypophysis.

Let us approach the problem from a somewhat different angle. If the pars anterior has a diuretic activity, it might be expected that extracts prepared from it would produce polyuria when administered to animals. There is adequate evidence to show that this does occur. Cushing and his coworkers[7] in 1910 augmented considerably the urine flow by the administration of a pars anterior extract to a dog with mild experimental diabetes insipidus. Numerous experiments[8] have been performed in which normal and mildly polyuric animals, including dogs, cats and rabbits were used; most of the experiments[9] have confirmed the presence of a diuretic effect from the administration of these extracts. However, there have been a number of negative results, chief among which are those of White[9] who has been unable to produce diuresis in normal rats with pars anterior extracts.

These experiments are difficult to evaluate. Extracts of the pars anterior which were mixtures of hormone fractions and various protein breakdown products have been used. Diuretic effects have been described by experimenters[8] who used acid extracts, alkaline extracts, salt suspensions, predominently growth factor, predominently thyrotropic factor and others. Precise experiments must wait until "pure" pituitary fractions are available for experimentation.

If we accept this body of evidence that there is a diuretic effect produced by the administration of pars anterior extracts, can we delineate any further the mode of action? It is reasonable that an action on water excretion might be the resultant of the many life processes controlled and maintained by the pars anterior of the pituitary. The changes that occur following ablation of the pars anterior are characterized by the loss of the various trophic functions with secondary atrophy of the thyriod, the adrenal, the gonads, and other organs. It is clear that the decreased metabolic exchange under these circumstances will be manifested by a small urine output and it is to be expected that replacement therapy with extract of the pars anterior will result in a return to a higher level of activity of the endocrine glands and a commensurate rise in urine output.

The search for a diuretic hormone is more specific than this. Is there evidence of a diuretic effect which can be explained by direct hormonal effect on the kidney function itself by an extract of the pars anterior? Or, is there possibly a direct effect by the thyroid or adrenals so that the pars anterior is indirectly involved through its trophic functions?

Several workers[10, 11] have recently emphasized the marked influence of the hypophysis on the kidney by demonstrating that the expected hypertrophy of the remaining kidney following unilateral nephrectomy does not occur in the hypophysectomized animal.

More recently, a number of careful and extremely important studies[12] have been made of the effects of extracts of the pars anterior or some specific renal functions. These studies were a logical follow up of some previous studies[13] on the effect of hypophysectomy on these same renal functions.

These experiments were performed on several types of animals which had been operated on. For our purposes the discussion will be limited to the dogs with ablation of the pars anterior. The clearance technics were used. Inulin clearance (glomerular filtration), diodrast clearance (effective renal blood flow) and diodrast-Tm (tubular mass) were determined. It was observed that the renal blood flow fell in five days to 50 to 60 per cent of the control level; the glomerular filtration paralleled this fall; the diodrast-Tm in the same space of time fell to 25 to 30 per cent of the control level. These effects were noted to last at least a year. A number of observations were made which indicated that these striking changes were not due to a fall in blood volume nor a fall in the blood pressure. It was reasoned further that these changes were not due to decreased metabolic rate since hypophysectomized dogs will take six to eight weeks to have a fall of 20 per cent in the metabolic rate. The effects above were apparent in less than a week and were of greater degree.

Studies were then made to determine whether administration of an extract of the pars anterior would be effective in returning these renal functions to normal in animals in which the pars anterior had been ablated, and also to determine whether normal dogs would have augmentation of these renal functions. The results were very striking; the increase of the effective renal blood flow (diodrast clearance), filtration rate (inulin clearance) and tubular mass (diodrast-Tm) was obtained in the normal dog; even more effect was noted in the animals in which there had been a depression due to removal of the pars anterior.

Unfortunately, no measurements were made of urine flow in these experiments. However, it is not unreasonable to assume that the urine flow paralleled the renal blood flow in these animals. These observations need verification, of course, and particular attention should be paid to the urine flow.

More precise studies will have to be made of cardiac output and the blood flow through organs other than the kidney in order to establish the specificity of the reported renal changes.

Is this reputed action of the pars anterior on kidney function mediated through one or several of the trophic hormones or is it a direct action of a "new" hypophyseal fraction? The answer is not available but there are a number of interesting facts which bear upon the question. The possibility that the described effect of the pars anterior is mediated through the thyroid has been given serious consideration. As described early in this paper, diuresis was produced in several types of animals by the administration of pars enterior extracts. It was discovered that these extracts did not produce diuresis in the absence of the thyroid gland[14]. It may be that the thyrotropic factor contributes to the diuretic effect but these studies of White and Heinbecker[15] demonstrate that the diuretic principle is not solely the thyrotropic hormone. It was shown that with a constant intake of thyroid insufficient in itself to produce diuresis, the thyroidectomized dog showed a diuretic response to pars anterior administration which was ineffective without thyroid. They suggested that in the dog thyroid might provide some sensitizing of the organism to the diuretic principle of the pars anterior or possibly thyroid might set the proper stage for pars anterior diuretic action by increasing the metabolic rate.

It would be most interesting to see the experiments of Heinbecker and White concerned with the changes in renal function (inulin clearance, diodrast clearance) performed in thyroidectomized and hypophysectomized-thyroidectomized dogs. It would be of great interest to know if the administration of pars anterior extract would produce any augmentation of the renal flood flow, etc. in the absence of the thyroid gland. These authors[12] have shown that thyroid administered to normal dogs and hypophysectomized dogs will result in augmentation of the renal blood flow and filtration rate similar to but not quite so large as occurs with pars anterior extracts.

In addition to the evidence presented in the preceding paragraphs, it is clear that the diuretic function of the pars anterior is not mediated solely through the thyroid gland via the thyrotropic hormone since total thyroidectomy does not cure diabetes insipidus. Findley and Heinbecker[16] reported that total thyroidectomy in a patient with diabetes insipidus diminshed the diuretic effect of sodium chloride and increased the antidiuretic effect of pitressin. It failed, however, to reduce urine flow below the limits set by a low salt diet.

Not too much needs to be said concerning the role of the adrenal as a diuretic force since one of the papers (Segaloff, A.: The Role of the Adrenal Gland in Sodium and Water Exchange) of this symposium is devoted entirely to the role of the adrenal in electrolyte control. There are several features of adrenal physiology which pertain especially to the problem on hand and should be mentioned.

Adrenal cortical extracts were studied for their effects on renal function (inulin clearance, diodrast clearance) parallel to the studies already mentioned[12] in which pars anterior extract and thyroid were used. It was found that inulin and diodrast clearances were unaffected or somewhat decreased by the administration of adrenal cortical extract to normal dogs and dogs with lesions in the hypophysis. The diodrast and inulin clearances, however, were increased in dogs in whom bilateral hypothalamic lesions had been produced. Diodrast-Tm was decreased in all the experimental dogs studied. These studies would tend to show at least that the effect of the pars anterior extract was not mediated through the cortical trophic hormone. The significance of the unique effect of the adrenal cortical extract in the hypothalamic puncture dogs is not sufficiently clear to discuss at the present time.

Studies of water diuresis have brought to light certain facts with reference to the adrenal cortex. There is evidence that the lack of normal water diuresis in hypophysectomized rats is due to the atrophic adrenals[17]. Water diuresis will not occur in hypophysectomized rats which are treated with pars anterior extracts (which will restore appetite growth) or thyroid (which will restore normal basal metabolic rate) or a combination of both. However, water diuresis is almost completely restored by relatively small amounts of adrenal cortical extract or desoxycorticosterone acetate. It is not clear whether this effect is mediated through a direct effect on the kidney or whether it represents the result of an effect on the shift of water between the cells and the extracellular spaces. Or conceivably, the adrenal may influence the release of the antidiuretic hormones, or, the sensitivity of the kidney to this hormone. Along the same line, and as poorly understood, should be mentioned the diabetes insipidus-like picture produced in dogs by the administration of very large doses of desoxycorticosterone acetate.[18] Finally, it should be mentioned that certain adrenal cortical fractions such as compound E in addition to an effect on carbohydrate metabolism tend to favor the excretion of salt and water[19].

The "diuretic role" of the adrenal requires considerable elucidation before it can be properly evaluated.

Is there a diuretic hormone? This paper has not produced the answer. If it has done anything, it has justified the asking.

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TURNOVER OF WATER AND SALT*

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Sodium and water are two of the most difficult of biologic substances to analyze and evaluate quantitatively. Because of this very little is known about their be- tion of tracer elements (Na^{24}, Deuterium) has partially increased our understanding havior in the human body. The introduc- but there is still much to be learned. This discussion is not an attempt to review the existing data, but is merely a summary of that which is of interest to the physician.

WATER

A 70 kilogram adult contains approximately a 50 liters of water divided into three compartments: the intracellular, containing about 35 liters; the interstitial, containing about 11 liters; and the vascular, containing about four liters.[1] Water taken in the gastrointestinal tract distributes itself throughout these compartments very readily and within an hour is in equilibrium with 60 per cent of the body weight.[2] Such water has an average stay in the body of approximately 14 days.[2] Normally it is taken in only through the gastrointestinal tract and lost from urinary, intestinal and respiratory tracts and through the skin. Of these routes of excretion, sweating is the most variable while diffusion through the skin is one of the most constant.

The rate of physical turnover in the body cannot be accurately measured but the information thus far available throws a great deal of light on such problems as edema formation. Nothing is known quantitatively of the exchange between the intra-cellular and interstitial compartments except the results of the studies of Darrow[3] These studies are concerned with fluid shifts resulting from electrolyte disturbance so are not applicable to this particular discussion (see below). The exchange between the vascular and interstitial compartments has been investigated by Flexner et al.[4] who find that 73 per cent of the blood water exchanges with extravascular fluid every minute. This is about three liters per minute or 4000 liters per day. From this exchange there is a surplus amounting to the lymph formed. This has been estimated as 40-50 liters per day or 99 per cent returned with 1 per cent as lymph. This same relationship is approximated by the kidneys in that 180 liters of water are passed into the glomerular filtrate with 1500-1800 c.c. ex-

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creted as urine. Approximately 10 liters are exchanged in the gastrointestinal secretions per day.

When water is considered as a chemical compound we find an additional exchange taking place which is of the same magnitude as the physical turnover. Barbour[2] states that one-third of the hydrogen atoms of protein and carbohydrates are in exchange with hydrogen atoms of water. What this means in terms of body water is not as yet apparent but the identity of water as a simple solvent must be reconsidered.

SODIUM

There are approximately 50 grams of sodium in a 70 kilogram adult. This is divided between two compartments instead of three as is the case with water, 34 grams in interstitial compartment and 15 grams in plasma. Little if any is contained within cells. Normally from 3-10 grams are taken in each day. An equal amount is excreted. The average life of sodium in the human body is not known with any certainty because the tagged molecules (Na^{24}) have an average life of only 21.2 hours. A longer life isotope is expected to yield information on this point. When taken in the gastrointestinal tract it appears quickly in the blood and urine. It is distributed only in the extracellular fluid of the body (20-25 per cent of body weight.[5]) In man it exists in concentrations of approximately 145 milliequivalents per liter in blood and interstitial fluid. When this value is exceeded it can mean one of two things: (1) Actual increase in sodium of body with corresponding increase in water; or (2) same amount of sodium but reduced amount of water for solution. The converse is true for lower values. These relationships must be kept in mind in evaluating blood sodium levels.

Sodium is not exchanged between the intracellular and interstitial compartments to any measurable extent. Gellhorn et al.[6] state that 56 per cent of plasma sodium diffuses through capillaries per minute. This is equivalent to 11 kilograms per day. The transfer in the kidney differs because sodium is retained but if calculated on the basis of glomerular filtrate concentration and reabsorption per cent (99.6)[1] it can be seen that approximately 900 grams are filtered through and 896 grams reabsorbed per day. Sodium is secreted into the gastrointestinal tract in concentrated form and by the sweat glands in a dilute form compared to plasma levels. In view of these figures it can be seen that imperceptible changes from the normal can produce marked changes in internal environment.

Considered chemically sodium is a very active ion and is exchanging constantly with similar ions in other sodium containing molecules ($Na\ HCO_3$, NaH_2PO_4, $Na_2\ HPO_4$). In addition there is another exchange not usually considered. Hodge et al.[7] have shown that approximately 21 per cent of the body sodium is combined with bone calcium (adsorbed to the hydroxyapatite crystal). This combination is one that readily takes up sodium from the fluid surrounding it when the molecular ratio with calcium is less than 1-30 and will release it slowly when this fluid becomes depleted. Such a mechanism provides a valuable reservoir in the body. The magnitude of 21 per cent of total sodium must modify current concepts of sodium balance.

Perhaps Na^{22} with its long average life of four and a fourth years will help to solve some of the problems of importance in sodium balance in health and disease.

In conclusion a brief outline of the effect of sodium depletion and excess will be given.

1. Marked deficiency of sodium (McCollum low sodium diet 00.2 per cent Na). Results: There is increased excretion of sodium when the intake is increased. This is similar to adrenalectomized animals and is prevented by cortical extract.

2. Moderate to borderline deficiency results in a retention of sodium greater than normal.[5]

3. A marked increase in sodium intake (20 gm./day for three days), results in excretion of a part of the sodium and an increase in the extracellular fluid compartment with normal re-established in three

days. When it is stopped diuresis starts in 12 hours and in 24 hours equilibrium is back to normal in regards to sodium and water (Darrow[3]).

4. An increase in sodium excretion caused with NH_4Cl being given for three days results in salt and H_2O both being lost with a decrease in the extracellular compartment. Normalcy is established in three days (Darrow[3]).

5. Adrenalectomized rats show an increased total sodium excretion including sodium added to diet; reverse is true of potassium. If sodium is available in moderate quantities (1 per cent in drinking water), sodium and potassium excretion becomes normal.[9]

CONCLUSION

As is true with any investigation of fundamental processes the addition of information such as this serves primarily to raise more questions and reveal more acutely a need for more information.

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WATER AND SODIUM BALANCE IN CONGESTIVE HEART FAILURE[*]

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That the mechanism of congestive heart failure is not fully established is generally agreed. Many clinicians, nevertheless, accept the ideas of "forward" and "backward" failure with a sense of satisfaction and complacency and fail to make any serious attempts to question them. There are many data which cast strong doubt on them. Unfortunately these data, though sufficient to question the validity of the classical concepts, are insufficient to establish a satisfactory explanation for congestive heart failure. Since congestive heart failure is so common and the problems of edema so important, it is the purpose of this presentation to summarize briefly some of the data found difficult to explain on the two generally accepted theories of congestive heart failure and to present a different concept.

Before entering into a discussion the two classical concepts of the mechanism of congestive heart failure are summarized.[1] The earlier of the two, "Forward Failure Theory," is based upon the idea that cardiac output is decreased and is, therefore, often referred to as the "Decreased Cardiac Output Theory." According to this theory the decrease in cardiac output results in anoxia in the tissues. This is followed by an increase in capillary permeability and edema with the classical clinical picture. This theory requires therefore that:

1. The cardiac output must be decreased for the period of the congestive heart failure syndrome.

2. The cardiac output must increase before improvement occurs, and

3. States which decrease the cardiac out-

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put must increase the severity of the congestive heart failure.

In the "Backward Failure Theory," it is stated that overwork of the heart leads to dilatation of the ventricle with the accumulation of blood because of a decrease in cardiac output (probably for only a few beats), an accumulation of blood in the veins with a resultant increase in venous pressure and venous blood volume. This is followed by an increase in intracapillary, hydrostatic pressure, circulatory stasis, capillary endothelial anoxia and an increase in capillary permeability with the resultant edema and classical picture of congestive heart failure. This theory would necessitate at least:

1. A decrease in cardiac output at some time,
2. High protein concentration in the edema fliud, and
3. Increase in venous pressure before the onset of congestive heart failure.

There are many observers who believe that both theories are required to explain the clinical picture of congestive heart failure.

In cardiac edema, there is an increase in volume of all of the fluid compartments, that is, (1) volume of the blood, (2) volume of the intercellular fluid, and (3) volume of the intracellular fluid. The intercellular fluid is isotonic, at a pH of 7.4, essentially normal in electrolyte concentration and relatively low in protein concentration (0.5 gram per 100 c. c.)

FACTS DIFFICULT TO EXPLAIN

There are many bits of data which make one question seriously the entire validity of the two classical concepts. Many of these are well known to every clinician, but in many instances these have never been considered collectively as serious objections to the two theories. These are:

1. Ligation of a large vein, the inferior vena cava just below the renal veins, will result in venous pressure values of 600 or more m.m. of water pressure in the tributaries without the development of clinical edema in the areas drained. It may be said that this is seriously different from the venous hypertension of congestive heart failure in that the renal circulation and renal function are not disturbed. This objection is over-ruled by the fact that in concretio cordis or mediastinal vena cava obstruction there is often marked venous hypertension for prolonged periods without clinical edema. In these states the renal circulation is distributed.

2. The intracapillary pressure in the capillaries of the feet increases to high levels when an individual stands erect. This pressure is often greater than the intra-arterial pressure in the arteries of the arms. Still clinical evidence of edema is not present. The tall man is no more prone to develop edema of the feet on standing than a short person. Therefore, the role of hydrostatic pressure in cardiac edema is very difficult to evaluate and is certainly not of the great value it is claimed to be.

3. There is no clinical edema in many severe anoxic states such as in tetralogy of Fallot or at high altitudes. With this anoxia, more severe than in congestive heart failure, there is no evidence of increase in capillary permeability, with edema formation. Such findings as these make it difficult to understand the importance of anoxia as a factor producing edema in congestive heart failure.

4. Patients with heart disease often gain weight before there is any increase in venous pressure. This again reduces the importance of increase in venous pressure or hydrostatic pressure in the explanation of cardiac edema.

5. Patients with heart disease who gradually and progressively go into congestive heart failure have been shown to present a drop in cardiac output without any rise in venous pressure when in Functional Class II. This is difficult to explain by either the "Forward" or "Backward" theories. Furthermore, when the failure progresses to the Functional Class III phase there was observed a simultaneous increase in both the cardiac output and venous pressure, findings which would be expected in view of Starling's law of the heart.[2] The re-

lationship of increase in venous pressure to cardiac output fails to abide by expectations planned by the two classical theories. Incidentally, the cardiac output has been found to be either decreased, increased or unchanged in patients with congestive heart failure.

6. The low protein content of the cardiac edema fluid, 0.5 gram per 100 c.c. is also difficult to explain by either of the two theories. If there was an increase in capillary permeability from anoxia there would necessarily be an increase in the concentration of protein in the edema fluid. The low protein content reduces the importance of capillary wall anoxia and increase in capillary permeability as a factor in the edema formation.

7. Extensive burning of the right ventricle of the dog with almost complete destruction of the entire myocardial tissue (checked by histologic sections) failed to be followed by a pattern of congestive heart failure.[3] Ligation of the coronary arteries to the right ventricle of the dog also failed to produce congestive heart failure. These observations are rather difficult to interpret in the light of the two classical theories.

8. The plasma proteins are usually normal, but may be elevated and are not usually low. Therefore, the edema and picture of congestive heart failure cannot be explained by changes in colloid osmotic pressure.

9. There is a decrease in cardiac output with venesection followed by clinical improvement. This is difficult to accept on the basis of the "forward" failure theory. With a decrease in cardiac output there should follow an increase in the severity of the failure.

10. A low salt diet is often followed by an improvement of the patient in congestive failure. Such a diet has no direct effect on the heart. This is difficult to explain satisfactorily by the two classical theories.

11. An over-does of desoxycorticosterone results in a clinical picture which is similar to that of congestive heart failure. This picture develops without there being any known direct cardiac change responsible for the syndrome.[4]

FINDINGS WHICH SUPPORT ANOTHER NEGLECTED MECHANISM

For many years there have been a gradual accumulation of data supporting a renal mechanism for most if not all of the syndrome of congestive heart failure. The idea that the kidneys may play an important role in congestive heart failure is not a new one. Baynard as early as 1722 suggested the idea.[5] Furthermore, it has been well known for many years that congestive heart failure is associated with an abnormal urine. There may be hematuria, albuminuria, many casts, a decrease in phenolsulfonphthalein excretion, and azotemia. These changes may reach high levels of abnormalities at times. They are usually casually referred to as being result of the "congestion of the kidneys." Little attention has been given to these changes as possible renal expressions suggesting a renal mechanism for the heart failure.

Before entering into a discussion of some of the newer data supporting a renal factor in heart failure it is well to point out three definite facts. There may be a possible exception to the first.

They are:

1. The initiating factor in the clinical syndrome of congestive heart failure is in the heart.

2. When a patient first sees his physician all of the accumulated fluid and electrolyte entered through the gastrointestinal tract. and

3. The renal excretion of salt and water lagged behind the intake, that is, at some time the patient was in a positive balance.

Therefore, by virtue of fact three above, regardless of the mechanism the kidneys have certainly failed to excrete enough salt and water.

DATA IN SUPPORT OF A RENAL MECHANISM FOR CONGESTIVE HEART FAILURE

There are certain observations which indicate a renal role in the congestive heart failure syndrome. These data are not complete and are so inadequate that no conclusions can be reached at present. They are sufficient, however, to support a need for careful study of the relationship of renal function to heart failure. The findings are:

1. The well known marked diuresis and improvement of a patient with congestive heart failure treated with mercurial diuretics show how effective a change in renal function can influence the heart failure syndrome. There is no known direct effect of the diuretic on the heart, the improvement being initiated elsewhere. The general impression is that the action is mainly through the renal tubules, although there is much good evidence to indicate an effect on protein fixed water and a possible action by way of the antidiuretic hormone of the posterior pituitary.

The diuresis produced by the mercury shows that enough glomerular filtrate is being formed in heart failure but that tubular reabsorption is too great. A reduction in the rate of tubular reabsorption results in a satisfactory loss of water and electrolyte with improvement of the patient. After all, 150 to 250 liters of glomerular filtrate are formed every 24 hours, while all of this is reabsorbed except for one or two liters. In congestive heart failure, the per cent change in filtration rate is relatively small while the per cent change in tubular reabsorption is great. It is quite likely that the latter is of great importance in the production of the congestive failure syndrome. It is well to remember that mercurial diuretics function very well in almost any type of edema.

2. Patients with congestive heart failure show a decided impairment of NaCl excretion following intravenous injection of 24 grams of sodium chloride.[6] The injection in normal subjects was followed by an excretion of 138-169 m. eq. of sodium per 24 hours while patients with congestive failure lost only 21-32 m. eq. in the same time. Not only was there less total loss but the concentration was less. Sodium and chloride parallelled each other. A mercurial diuretic increased the sodium excretion rate from about 30 m. eq. per 24 hours to 107. The concentration also increased. These changes were not apparently due to changes in rate of glomerular filtration as the urea clearance remained unchanged by the failure.[6]

These studies unfortunately did not include a variety of non-cardiac edema states. The above findings may not be peculiar to cardiac edema.

By what mechanism congestive failure disturbs renal function is not known. The role of the hormones, adrenal cortical and antidiuretic, can only be conjectured. It is possible to formulate a fairly satisfactory theory on the basis of disturbances in their function.

The studies of Warren and Stead[7] in which they produced "congestive heart failure" by oral administration of 12 grams of NaCl per day to three patients with heart disease and on the verge of failure fails to prove a renal cause of congestive heart failure, in that there was no control group of patients with other types of edema nor has one the right to refer to such a precipitated edema as being an example of congestive heart failure. Their work casts considerable doubt on the two classical theories.

3. The studies of Starr[8] with the use of a model of the circulation failed to support the two classical concepts of congestive failure. Such non-physiologic studies cannot be taken too seriously without physiologic data of direct sort to support it. The studies of "static blood pressure" in patients with congestive heart failure and patients without failure or heart disease shortly after death showed that heart failure was associated with an increase in blood volume and venous pressure when the heart action had ceased. Starr's studies unfortunately did not include patients with various types of non-cardiac edema. Starr suggested from his studies a possible renal factor in cardiac edema.

4. Seymour et al.[9] found the inulin clearance (rate of glomerular filtration) to be normal in congestive heart failure. Phenol red clearance (rate of effective renal blood flow) was invariably less than nor-

mal. The reduction in effective renal blood flow was proportional to the decrease in cardiac output. They failed to study patients with non-cardiac edema. These observers considered the data to indicate a greater rate of tubular reabsorption than in the normal. Furthermore, their data indicate a great filtration pressure since more glomerular filtrate was formed from less blood.

Merrill et al.[10] found, by direct catheterization of the renal veins, both the inulin and diodrast clearance to be reduced in congestive failure. The per cent reduction in the rate of renal blood flow was greater than the per cent reduction in the cardiac output. These observers considered the data to indicate an increase in filtration pressure and therefore constriction of the efferent glomerular arterioles. Bioassay showed a great increase in renin in the blood of the patients with congestive heart failure. No data were collected in patients with non-cardiac edema.

5. Schemm's[5,11] method of restricting sodium chloride and forcing water or the more conventional method of restricting sodium and allowing water libidum results in improvement of congestive failure. This apparently is not associated with direct cardiac action. It is quite likely to be closely associated with renal function in the maintenance of electrolyte balance and isotonic extracellular fluids.

SUMMARY

The above data only show the need for more studies of the mechanism and physiologic state of congestive failure. In fact, it is very unlikely that patients have ever been studied during the period that the disturbances actually responsible for the syndrome were at work. It is quite likely that these disturbances are at work for only a few seconds or heart beats and only the resultant changes have been studied to date. Furthermore, when it is realized that over 4000 liters of water leave the blood stream and return from the tissues every 24 hours, it is easily visualized how subtle the changes can be and still result in the clinical syndrome. The present day methods are not sensitive enough to measure the changes that would be involved under such circumstances.

It is safe to conclude that the mechanism of congestive heart failure is unknown and a syndrome so common as this deserves greater research attention.

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THE ARMY DOCTORS

There is undoubtedly a feeling amongst the doctors who sacrificed everything to go into the army and navy to help their country during the last great war that their services have not been entirely appreciated. They feel that the difficulties that have arisen in attempting to get their former practices established have been indeed great and that not much sympathy has been given to them in their endeavors nor has there been entirely a true appreciation of their unselfish call to duty.

It is hard to convince many medical officers that we who stayed at home do, with all our heart, feel grateful to them for what they have done. All this is apropos of the celebration of the 157th anniversary of the establishment of the medical department of the U. S. Army when General Eisenhower, in a coast to coast radio broadcast, praised the medical department for its great successes before and during World War II.

Another appreciation of the army medical services which were rendered practically entirely by the volunteer practicing physicians of this country is a recent editorial in Collier's, for August 3, written by Dr. Louis I. Dublin, entitled, "The Medics in World War II."

We think that this editorial in Collier's also proves that we are indeed thankful to the doctors for what they have done. The editorial is well worth reproducing.

"Casualty and disease statistics on World War II have been pretty well rounded up and collated now; and out of the statistical picture the U. S. medical profession emerges with what can only be described as glory. This applies particularly to the physicians who went to the war and took care of the wounded and dying.

"Our combat deaths, according to the Metropolitan Life Insurance Company's latest estimate, amounted to a little more than 300,000. Up to V-J Day our deaths from disease came to about 20,000, or 1-15th of the combat total.

"This was in striking and consoling contrast to our experiences in all previous wars. About the most harrowing reading you can find in connection with the Civil War is an occasional description of hospital filth, disease, and the miseries of the wounded, most of whom would literally have been better off dead. In the Spanish-American War, only 48 years ago, we lost about 15 times as many men from disease as we lost in combat—the exact reverse of the World War II ratio. In World War I, only 28 years ago, our deaths from disease totaled slightly more than those from enemy action.

"Yet in the second great war, our fighting forces were flung much farther afield and into more fearful and wonderful climates than they had encountered in any of our other wars. They fought through malarial jungles in the South and Southwest Pacific, through filthy North African towns and rural areas, and in many a country or on many an island where sanitation was felt by the natives to be just a rather dull joke.

"Yet so highly organized and generously supplied were our medical services, and so devoted were our doctors and nurses in the field, that disease and infection were cut to the proportions of a minor menace. The percentage of wounded who died of their wounds, too, was reduced to the record low of 4 per cent, as contrasted with almost 8 per cent in World War I.

"There is only one moral to all this that we can think of. It is: Let's just keep pounding away at public health activities of all kinds, and at implementing the medical profession for more and faster progress in the conquest of disease. Our long zeal in these directions paid tremendous dividends in our latest war."

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STATE HEALTH OFFICER

We were very much surprised to hear that Dr. David E. Brown was not reappointed as head of the State Board of Health. The state health officer is virtually a member of the governor's cabinet and it is certainly unusual that a recommendation made by the governor for that particular position is not approved by the senate. The only explanation that has been offered is that politics were eminently concerned with this appointment. No criticism has been made of Dr. Brown as a state health officer and certainly he has gotten along well for the most part with the medical profession in the state. It may be true that occasionally a minor difference of opinion arose between Dr. Brown and individual physicians but on the whole his tenure of office has been marked by a desire on his part to cooperate with the medical profession. It seems a remarkable thing that a man who has proved himself and has shown that he is capable of conducting a state health department should be turned down because a few senators have gotten together and have agreed among themselves that certain reappointments should not be made. It seems that there was a block who decided that they would not recommend certain of Governor Davis' appointments. As far as we can learn there were only two or three senators opposed to Brown but their feeling was that if they would in turn agree not to vote for the approval of someone else that they could block the appointment of an efficient state health officer. This was apparently accomplished. It seems like awfully picayunish and petty politics. Dr. Brown should be commended on not being amenable to pressure on the appointment of someone to a minor position who apparently was not qualified but yet had the good fortune to be a relative of a senator.

The State Board of Health definitely should be out of politics. The importance of politics in this position may be exemplified by the fact that the previous incumbent to the office was willing to give up his position, in fact he became desirous of doing so in order that he might qualify under civil service regulations and be assured of his future in the State Board of Health.

It is indeed fortunate that there was available a man in the Department who was well qualified and who will be an excellent state health officer but he, who is a career man in public health, may feel before long that political pressure may be such that it would be better for him to be under civil service than to head this important department.

By far the most important implication is the fact that the medical profession of the state of Louisiana does not have an active role in the appointment of the state health officer. The state health officer should be appointed on the basis of a recommendation or recommendations by the State Medical Society. Certainly in our two neighboring states whose health officers have been outstanding, maintenance of their position has depended entirely on the medical profession. The late Dr. Baker of Alabama who was appointed and kept in office by the

State Medical Society, is representative. Dr. Underwood of Mississippi, one of the best known health officers in the country, likewise has had a long tenure because of the state medical profession and their whole-hearted backing-up of this gentleman.

The Louisiana State Medical Society should have a definite say as to who is to be the state health officer and should have more power than they have in important medical appointments.

THE SYMPOSIUM

In this number of the Journal there is being published a symposium on salt and water balance. The papers that appear in this symposium are somewhat unusual for the Journal. It is not as a general rule that this type of presentation is published, however it is felt that the physician who has the time and the inclination to read the papers that appear in the symposium will derive much information, which is up to the moment from the laboratory and clinical studies of body fluids which have been made in recent months and years. The papers may seem too scientific for a journal essentially clinical but the reader must bear in mind that there are extremely important clinical implications in these presentations. Edema is a common symptom; it is discussed in its relation to salt metabolism. The use of diuretics is stressed and there is presented a discussion of the hormone control of water and salt. Of course of extreme importance to the doctor is the relationship of heart failure to clinical edema and renal excretion of salt and water. The fundamental knowledge of these data will do much to enable the doctor to treat his patient with a clear understanding of the purposes and the mechanism concerned when he outlines the course of therapy for the edematous patient.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

MESSAGE FROM THE PRESIDENT

The most important activity of organized medicine at this time is to continue the fight against the Wagner-Murray-Dingell bills and similar legislation tending toward socialization of medicine. Although S.1606 did not come out of committee at the last session of Congress hearings will no doubt be continued at the next session or a third edition of the bills presented. It is thought that the medical profession at this time should take advantage of the lull and prepare for the next fight which is sure to come. Among the aims here should be to educate the public as to the views of the medical profession on such bills and to establish a better relationship with the public informing them of the results of such legislation to the medical profession and to the general public. It is felt that this should be one of the main objectives of the State Society at this time.

Special announcement concerning plans for prepayment medical service is included in this section of the Journal. It is hoped now that such a service may be put into effect by October 1. Dr. O. B. Owens and his committee have been directing their efforts to this accomplishment and we feel reasonably certain that plans will be completed by that time.

A third project before the organization is to effect a program for medical service to veterans in cooperation with the Veterans Administration, whereby the veterans may obtain medical care in their home communities by home-town doctors. Detailed plans for such service will possibly be completed within a very short time and all members of the Society will be appropriately notified concerning regulations for participation in the plan.

Efforts are being made by the present administration to improve the scientific program for the annual meeting so that every member wil be given a worthwhile reason for attending the meeting next year. Many suggestions have been considered and announcement will be made when details have been worked out.

If these four activities—(1) defeat of bills advocating socialization of medicine; (2) prepayment medical service; (3) medical service for veterans; (4) improved scientific programs—are consummated it is felt that the present administration will have some claim of having been moderately successful.

LOUISIANA PHYSICIANS SERVICE ORGANIZED

The organization of a voluntary prepayment surgical and obstetrical care plan insurance company in the State of Louisiana is announced by Dr. Rhett McMahon, President of the State Society. The new company, sponsored by the Louisiana State Medical Society, was incorporated August 18, 1946 and is known as the Louisiana Physicians Service, Inc., and will operate on a statewide basis.

Louisiana Physicians Service is designed primarily to relieve the family of average means, particularly those of the low income group, from financial distress that frequently accompanies prolonged or catastrophic illness, but it is also available to the more fortunate as an attractive investment and a convenience.

Participation is voluntary. It leaves unaltered the established custom in the practice of medicine and in no way interferes with the individual's selection of a physician. Louisiana Physicians Service is a nonprofit mutual corporation organized by the doctors of the State of Louisiana and dedicated to the people of the State of Louisiana.

The organization of the company is the result of over two years' work by members of the Louisiana State Medical Society's Committee on Prepayment Medical Service, with Dr. O. B. Owens, Chairman.

Appointed to represent the Louisiana State Medical Society on the Board of Trustees are the following: Drs. Rhett McMahon, Baton Rouge; O. B. Owens, Alexandria; Wm. L. Bendel and George Wright, Monroe; H. Whitney Boggs, J. P. Sanders and M. D. Hargrove, Shreveport; A. V. Friedrichs, E. L. Zander, Val H. Fuchs, P. T. Talbot and Gilbert C. Anderson, New Orleans; U. S. Hargrove, Baton Rouge; W. P. D. Tilly, New Iberia; C. M. Horton, Franklin; and J. W. Faulk, Crowley.

Members of the new organization's Board of Directors are Drs. O. B. Owens, H. Whitney Boggs, Wm. L. Bendel, U. S. Hargrove, C. M. Horton, J. S. Faulk, E. L. Zander and Val H. Fuchs with Drs. Rhett McMahon and Gilbert C. Anderson as ex-officio members.

Dr. O. B. Owens was elected President of the newly organized company and Dr. Wm. L. Bendel, Vice-President, Dr. H. Whitney Boggs, Secretary-Treasurer and Frank Lais, Jr., Executive Director, compose the other officers.

All doctors of medicine licensed by the Louisiana State Board of Medical Examiners are invited to become participating physicians in the plan.

Louisiana Physicians Service does not contract to provide medical care nor may a participating physician be compelled to perform his services in any way differing from usual custom, except that the participating physician agrees to accept the fees in the Schedule of Benefits, as approved by the Louisiana State Medical Society, as full payment for service rendered after determining with the subscriber that his yearly income does not exceed $1,800 if single or $2,400 if carrying a family membership.

There is no "red tape" attached to the physician's request for payment for services rendered and the physician is assured of receiving his fees under the plan, particularly fees due from members of the low income group.

The physician will make his usual charge to patients in the "indemnity" classification as distinguished from the members of the low income group. Patients in the higher income group will pay that portion of the fee, if any, that remains after payment has been made as an indemnity by the company to the physician.

There are some fifty-three plans, ranging from local to statewide, in operation at the present time. The total enrollment exceeds 2,800,000 persons. The number of units and subscribers has been growing since 1939. So far this year, doctor-endorsed plans have been introduced in eight states.

The recent spread has coincided with a controversy over methods of handling the problem of paying doctors' bills. President Truman has proposed a national system of compulsory prepaid health insurance to cover the cost of medical, dental, surgical and hospital care. The American Medical Association, contending such a setup would constitute "socialized medicine", has set out to aid the development of voluntary, prepayment plans sponsored and approved by state and local medical societies.

It is expected the Louisiana plan will begin operation on October 1, 1946. Doctors throughout the state will subsequently be contacted relative to their participation in the plan.

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MEDICAL SERVICE COUNCIL NAMED

The appointment of Dr. A. V. Friedrichs, chairman; Dr. O. B. Owens, Dr. U. S. Hargrove, Dr. John G. Snelling and Dr. P. T. Talbot as members of the Council on Medical Service and Public Relations in the Louisiana State Medical Society was announced recently by Dr. Rhett McMahon, President of the Louisiana State Medical Society.

Frank Lais, Jr., has been named as the council's Executive Director.

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TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

Sept. 11.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Sept. 12.	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Sept. 18.	The Orleans Society of X-Ray Technicians, 7:30 p. m.
Sept. 25.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Sept. 26.	Clinico-pathologic Conference, Touro Infirmary, 12 noon. DePaul Sanitarium Staff, 8 p. m.
Sept. 27.	L. S. U. Faculty Club, 8 p. m. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Oct. 1.	Eye, Ear, Nose and Throat Staff, 8 p. m. Orleans Parish Radiological Society, 7:30 p. m.
Oct. 2.	Mercy Hospital Staff, 8 p. m.
Oct. 3.	Executive Committee, Baptist Hospital, 8 p. m.
Oct. 4.	Ochsner Clinic Staff, 8:30 p. m.
Oct. 7.	Board of Directors, Orleans Parish Medical Society, 8 p. m.
Oct. 8.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
Oct. 9.	Woman's Auxiliary, Orleans Club, 3 p. m. Touro Infirmary Staff, 8 p. m. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Oct. 14.	Scientific meeting, Orleans Parish Medical Society, 8 p. m.

Every Wednesday, Medical Discussions, Radio Station WDSU (1280 on your dial).

MAX M. GREEN, M.D., Secy.

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Dr. Charles J. Bloom recently addressed a meeting of the Pike County Medical Society in McComb; his subject was "Rheumatic Fever." On July 3 Dr. Bloom was the guest speaker at a meeting of the Gulf Coast Medical Society in Biloxi; he spoke on "Recent Progress in Pediatric Treatment."

At present Dr. Bloom is in Saluda, North Carolina, teaching at the Southern Pediatric Seminar.

Drs. Joseph A. Danna, Val Fuchs, and C. Walter Mattingly were recently appointed as a medical advisory committee to the American Red Cross. Dr. Danna will serve as chairman of the committee.

Dr. F. L. Jaubert recently addressed the conference of the National Foundation for Infantile Paralysis on the problem of crippled children in the state of Louisiana.

Dr. Edward W. Nelson recently presented a paper on "Phlebothrombosis and Thrombophlebitis in Obstetrics and Gynecology" at the Rapides Parish Medical Society in Alexandria. Following the presentation a colored movie outlining the technics of vena cava ligation was shown.

At a recent meeting of the Seventh Congressional District Medical Society in Jennings, Dr. H. Randolph Unsworth gave a paper on "Psychosomatic Medicine" and Dr. Samuel B. Nadler spoke on "Surgical and Nonsurgical Jaundice."

Dr. G. J. von Langermann was elected treasurer of the Altrusa Club at a meeting of this organization held July 17.

WELCOME HOME

The following physicians were recently separated from military service: Louis J. Bristow, Jr., Baptist Hospital; Richard Buck, Audubon Bldg.; Mercer G. Lynch, Maison Blanche Bldg.; Hardee Bethea, 1007 American Bank Bldg.; M. Lyon Stadiem, 1201 Canal Bldg., Roy H. Turner, 1430 Tulane Ave.; Maurice W. Geldert, Jr., Southern Baptist Hospital.

CALLED TO MILITARY SERVICE

The following intern members were recently called to military service: Hiram W. K. Batson, Richard A. Faust, George E. McCaskey, William L. McLeod, Joseph L. Rosenzweig, George T. Schneider, William H. Syll, Gerald N. Weiss, and Earl E. Wilkinson.

CHANGES IN MEMBERSHIP ROLLS

During the month of July the following changes took place in our membership rolls:

Three physicians were transferred to the active membership rolls: Drs. Robert B. Haspel and Horace L. Wolf, former intern members, and George D. Berkett, formerly an associate member.

Five physicians removed from New Orleans and were removed from the rolls—Drs. Robert M. Rose (active), Arthur A. Calix, Eugene Hesdorffer, Pedro Suau (intern), and Harry C. Knight (associate).

CHANGES OF ADDRESS

Dr. Luis R. Oms, 505 Legendre Bldg.; Albert Segaloff, Ochsner Clinic; Wm. H. Block, 619 St. Philip St.; Dr. Henry D. Ogden, 1706 Pere Marquette Bldg.; W. P. Bradburn, Muir Bradburn, W. R. Brewster, R. J. Christman, Hyman L. Cohen, Paul McIlhenney, 1326 Foucher St.

RADIO PROGRAM

The following "Health Discussion" will be broadcast over radio station WDSU (1280 on your dial) during the month:

August 14—Eye Strain—Dr. John B. Gooch and Mr. Joseph M. Fallo.

August 21—Complications of the Diabetes Mellitus—Drs. Robert A. Katz and Mr. Fred R. Wulff, Jr.

August 28—Medical Ethics—Dr. Val Fuchs.

September 4—Prepayment Medical Plans—Mr. Edward Groner.

HANDY LITERATURE FILE

A sturdy cloth-bound literature filing box is being presented to all physicians by Schering Corp. of Bloomfield and Union, N. J.

Through direct mail, the physician receives many scientific folders designed to keep him well informed on the latest developments in the pharmaceutical field. Many physicians have desired a file box to preserve this valuable literature for ready reference. Designed to simulate a technical reference text and bound in red-purple cloth and stamped in gold, the handy file boxes are presented free of charge as another Schering service to physicians.

MEDICAL TOASTMASTERS

The Medical Toastmasters have adjourned for the summer. They will resume classes in the fall.

During this recess a canvass of the membership of the Society will be made to secure new members.

As previously mentioned these classes are held to improve one's oratorical ability. Dale Carnegie's textbook on public speaking is the basis of teaching; there are sixteen lectures in all. Each member of the class is called upon to give a three-minute talk, and is quizzed on certain sections of the night's lecture.

It has been suggested that when classes resume they be held in the Hutchinson Memorial Auditorium, 1430 Tulane Avenue, at 8 or 8:30 p. m., rather than having a dinner meeting at 6:30 p. m. at the New Orleans Athletic Club.

Another suggestion that has been made is that an English professor or a director of public speaking at one of the high schools or universities be engaged to correct delivery, diction, etc.

If you are interested in participating in these classes you are urged to communicate with Dr. Edwin L. Zander, president of the Medical Toastmasters, or Dr. L. J. O'Neil, the secretary, as there is a limit to the number of members that these classes may accommodate.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

ATTENDANTS AT A. M. A. MEETING

The following members of the State Medical Society attended the annual meeting of the American Medical Association which was recently held in San Francisco. From New Orleans:

Drs. Hartwig M. Adler, Ansel M. Caine, Curtis W. Caine, Charles L. Cox, Jules Myron Davidson, M. E. DeBakey, Thomas Findley, Warren Hebert, J. C. Henthorne, M. O. Hines, James K. Howles, Edgar Hull, Robert A. Katz, Maurice Lescale, Wilkes A. Knolle, Alton Ochsner, Walter J. Otis, Cassius L. Peacock, Ralph V. Platou, P. J. Saleeby, John H. Seabury, Daniel N. Silverman, Roy H. Turner, Woodard D. Beacham, George E. Burch, Guillermo M. Carrera, Boni J. DeLaureal, Emma S. Moss, William Parson, John C. Weed, B. Bernard Weinstein, Monroe Wolf, John Adriani, Donovan C. Browne, Eugene B. Vickery, E. Carroll Faust, and Champ Lyons.

From elsewhere in the state: Drs. W. P. Butler, A. A. Herold, C. P. Rutledge, J. D. Talbot, Shreveport; Drs. H. O. Barker, L. D. Gremillion, O. B. Owens, Alexandria; Dr. J. Q. Graves, Monroe; Dr. L. H. Lafargue, Sulphur; Dr. D. F. Davis, Lake Providence; Dr. S. F. Fraser, Many; Dr. H. M. Prothro, Pleasant Hill.

TULANE FACULTY MEMBERS COMMENDED

Six members of the faculty of the Tulane Medical School and one former member have been commended by the O.S.R.C. in conjunction with researches they carried out in cooperation with or at the suggestion of this committee. The members to be commended are Dr. Ernest Carroll Faust, professor of parasitology; Dr. Ralph G. Smith, professor of pharmacology; Dr. R. V. Platou, professor of pediatrics; Dr. Alton Ochsner, professor of surgery; Dr. Guy A. Caldwell, professor of orthopedics; Dr. George E. Burch, associate professor of clinical and experimental medicine, and Dr. Walter J. Horton, former assistant professor of chemistry, now head of the department of chemistry at the University of Kansas.

CONSULTANTS TO THE SECRETARY OF WAR

The Office of The Surgeon General announces the appointment of medical consultants throughout the United States. The following men have been selected from the south: Dr. Walter B. Martin, Norfolk, Va.; Dr. John Minor, Washington, D. C.; Dr. Henry M. Thomas, Jr., Baltimore, Md.; Dr. Roy H. Turner, New Orleans, La.; Dr. Wm. T. Rainey, Fayetteville, N. C.; Dr. Virgil P. Sydenstricker, Augusta, Ga.

MISSISSIPPI VALLEY MEDICAL SOCIETY

This organization will meet at the Jefferson Hotel, St. Louis, on September 25-27. A distinguished list of guest speakers have consented to appear on the program.

NEW JOURNAL OF DERMATOLOGY AND SYPHILOLOGY

The publication of a new journal on dermatology and syphilology, the Quarterly Review of Dermatology and Syphilology, has been announced by the Washington Institute of Medicine. The Editor-in-Chief of this new journal will be Donald M. Pillsbury with Herman Beerman and Clarence S. Livingood, associate editors.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examination (Part I) will be held on Friday, February 2, 1947. As a number of changes in Board regulations and requirements were made effective at the last annual meeting of the Board held last May, it would be advisable for candidates for licensure to communicate with Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh 6, Pennsylvania.

AMERICAN HOSPITAL ASSOCIATION

Rear Admiral Dallas G. Sutton (MC) USN (Rt.) comes to the Washington Service Bureau staff of the American Hospital Association on September 1 as director of study of government hospital relations. Admiral Sutton will serve as a liaison between the association and the federal government in matters concerning the federal hospital program and will assist in the coordination of civilian and federal hospital facilities.

Among Admiral Sutton's more recent assignments was that of Assistant Surgeon General, 1937 to 1941. During 1941 and early 1942 he was commanding officer of the Naval Hospital, Portsmouth, Virginia, after which he was in charge of construction of the Naval Medical Center at Bethesda, Maryland, erected the same year. From that time until his retirement in April of this year, Admiral Sutton was inspector of all east coast activities of the Naval Medical Department.

NEWS ITEMS

The seventh Annual Congress on Industrial Health sponsored by the Council on Industrial Health of the A. M. A. will be held at the Copley-Plaza Hotel, Boston, Massachusetts, September 30-October 2.

The National Society for the Prevention of Blindness will hold a three day conference at the Hotel Pennsylvania, New York, November 25-27.

Colonel Frank S. Gillespie of the Royal Army Medical Corps was presented the Legion of Merit by General Eisenhower. Innumerable medical men have had the opportunity of meeting and knowing Dr. Gillespie who served for some four years as British Medical Liaison Officer in the Army Medical Department.

NURSES FOR POLIOMYELITIS

On account of the marked increase in number of cases of poliomyelitis this year there is a dearth of nurses to take care of these children. The Red Cross is preparing to recruit nurses to be paid by the National Foundation for Infantile Paralysis in the states in which the disease is particularly rife. Louisiana is one of 11 states which requires additional nursing help.

PHYSICIAN WANTED

A cooperative insurance group recently organized in Pollock, Louisiana is desirous of securing services of a doctor; salary $200-$300 per month, office furnished. Outside general practice available.

For further information contact Mr. W. E. Groves, 420 Audubon Building, New Orleans. Telephone Ra. 6093.

TWENTY-FIFTH ANNIVERSARY OF THE DISCOVERY OF INSULIN

The twenty-fifth anniversary of the discovery of Insulin will be observed with a program in Convocation Hall, at the University of Toronto, on September 16. Many internationally known figures in the field of medicine will be present to honor the occasion. Among them will be R. D. Lawrence, physician in charge, Diabetic Clinic, Kings College Hospital, London, England; H. C. Hagedorn, of Gentofte, Denmark; Bernardo A. Houssay, Research Institute of Experimental Biology and Medicine, Buenos Aires, Argentina; and Elliott P. Joslin, Harvard Medical School, Boston, U. S. A. This observation will be followed by the regular annual meeting of the American Diabetes Association.

On September 23, Eli Lilly and Company will sponsor an International diabetes clinic to be held at the Indiana University Medical Center in Herty Hall of the State Board of Health Building, Indianapolis, Indiana. International importance will be given to this meeting by the presence of Professor Charles H. Best, Toronto, Canada, co-discoverer with Banting of Insulin, Professor Houssay, Dr. Lawrence, and Dr. Hagedorn. They will discuss various phases of diabetic care.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that for the week ending July 13 there were 148 deaths in the City of New Orleans, as contrasted with 106 the previous week. These deaths were divided 103 white, 45 non-white, with 27 of the total occurring in children under one year of age. For the week which came to a close on July 20 there was a considerable reduction in number of deaths of citizens of New Orleans; 124 people expired in this particular week, 80 of whom were white, 44 of whom were colored and only seven of the total being in children under one year of age. There was a slight increase in the number of deaths for the week ending July 27 but as in the past some months the number of deaths is below the three year average of the corresponding week of the year. In this particular week the 131 deaths were divided 82 white and 49 colored with 17 deaths in children under one year of age. For the week which closed August 3 the 128 deaths in the City of New Orleans were apportioned 95 white, 33 colored, with an astounding low infant mortality of only five.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending June 29 showed measles leading all other reportable diseases with 53 cases and only three other diseases occurring in numbers greater than ten, namely, pulmonary tuberculosis with 22 cases, poliomyelitis with 13 and mumps with 11. The very large number of poliomyelitis cases were confined almost entirely to the southern parishes of the state; Lafayette Parish with four cases, Iberville with three and East Baton Rouge with two, were the parishes which had more than one case. For the week which closed July 6 the following diseases occurred in numbers greater than ten; cancer 173, unclassified pneumonia 114, measles 57, mumps 50, malaria 45, rheumatic fever 35, pneumococcic pneumonia 33, chickenpox 28, septic sore throat 19, whooping cough 17, influenza 15, poliomyelitis 14, amebiasis 13 and hookworm infestation ten. Again, the poliomyelitis cases were found to have occurred largely in the southern parishes, Orleans and Vermilion with three each, were the only two parishes which had a greater number than one case. All of the malaria cases were contracted within the Continental United States. For the week which closed July 13 pulmonary tuberculosis was reported in 78 instances, followed by 18 cases of poliomyelitis, 14 of typhus fever, 12 of unclassified pneumonia and ten of measles. The poliomyelitis cases, except for two reported from Caddo and one from Ouachita, were again confined to the parishes in the southern portion of the state.

Indicative of the presence of poliomyelitis is the fact that for the week ending July 20 it was second in the list of reportable diseases, with 27 cases, preceded by pulmonary tuberculosis only, with 29 cases. Other diseases in numbers greater than ten included 20 cases each of malaria and typhus fever, 17 of diphtheria, 11 each of measles and typhoid fever and ten of scarlet fever. The poliomyelitis cases were reported in greatest number in Ouachita Parish with five instances, followed by Orleans Parish with three, Caddo with two and East Baton Rouge with two. Other reported cases were found in numbers no greater than one in various parishes throughout the state. For the week ending July 27 there were listed 44 cases of pulmonary tuberculosis, 35 of measles, 18 each of poliomyelitis and of whooping cough, 15 of unclassified pneumonia, 13 each of septic sore throat and influenza and 11 of mumps. There was a reduction in the number of cases of poliomyelitis reported this week but the number is still large. So far up to date this year 148 patients with poliomyelitis have been reported to the State Board of Health. Of the 18 cases this week no one parish had more than two originating within its borders.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending June 30, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	54	284	338
Gonorrhea	1345	8837	10182
Granuloma inguinale	25	143	168
Lymphopathia venereum	12	55	67
Syphilis	1651	8402	10053

PHARMACOPEIA REVISION

Many requests have been received by the Committee on Revision of the Pharmacopeia of the United States of America relative to the deletions which will be made in the U.S.P. XIII of titles representing items which were official in the U.S.P. XII. The list of drugs omitted is as follows:

Acetonum
Acidum Aceticum
Acidum Aminoaceticum
Acidum Lacticum
Acidum Mandelicum
Acidum Nitricum
Acidum Phosphoricum
Acidum Phosphoricum Dilutum
Acidum Sulfuricum
Acidum Sulfuricum Dilutum
Aethylis Chaulmoogras
Alcohol Dehydratum
Allylis Isothiocyanas
Althaea
Antipyrina
Aqua Chloroformi
Argentum Proteinicum Forte
Belladonnae Radix
Bismuthi Subnitras
Calcii Phosphas Tribasicus
Capsulae Carbonei Tetrachloridi
Capsulae Olei Chenopodii
Capsulae Triasyni B Cum Hepati
Carbonei Tetrachloridum
Ceratum
Ceratum Resinae
Chloramina-T
Codeina
Colchici Semen
Concentratum B-Vitaminarum Hepatis
Decocta
Emplastrum Belladonnae
Emulsum Olei Terebinthinae
Ergota
Eriodictyon
Eucainae Hydrochloridum
Extractum Hyoscyami
Extractum Malti
Extractum Rhei
Fel Bovis
Ferri Et Ammonii Citrates Virides
Ferrum Reductum
Fluidextractum Ergotae
Fluidextractum Eirodictyi
Hydrargyri Bichloridum
Hydrargyri Salicylas
Hydrargyri Succinimidum
Hydrargyrum cum Creta
Infusa
Injectio B-Vitaminarum Hepatis
Injectio Hydrargyri Salicylatis
Injectio Strophanthini
Injectio Triasyni B cum Hepati
Linum
Liquor Acidi Arseniosi
Liquor Hydrogenii Peroxidi Fortior
Liquor Iodi
Lycopodium

Magnesii Phosphas Tribasicus
Massa Ferri Carbonatis
Mel
Nux Vomica
Oleum Amygdalae Amarae
Oleum Chaulmoograe
Oleum Chenopodii
Oleum Juniperi
Oleum Lini
Oleum Picis Rectificatum
Oleum Pini Pumilionis
Oleum Terebinthinae
Oleum Terebinthinae Rectificatum
Pamaquinae Maphthoas
Pelletierinae Tannas
Phenylis Salicylas
Pilulae Ferri Carbonatis
Potassa Sulfurata
Potassii Bitartras
Potasii Nitras
Pulvis Cretae Compositus
Quininae Aethylcarbonas
Quininae et Ureae Hydrochloridum
Resina
Serum Antimeningococcicum
Serum Immune Morbillosi Humanum
Serum Immune Scarlatinae Humanum
Sevum Praeparatum
Sodii Cacodylas
Spiritus Anisi
Spiritus Camphorae
Spiritus Frumenti
Spiritus Glycerylis Trinitratis
Spiritus Vini Vitis
Strophanthinum
Sulfapyridinum
Sulfapyridinum Sodicum Sterile
Syrupus Picis Pini
Tabellae Magnesii Phosphatis Tribasici
Tabellae Sulfapyridini
Terpini Hydras
Thymolis Iodidum
Tinctura Colchici Seminis
Tinctura Lodi (7 per cent)
Tinctura Nucis Vomicae
Toxitabellae Hydrargyri Bichloridi Magnae
Toxitabellae Hydrargyri Bichloridi Parvae
Trinitrophenol
Unguentum Iodi
Zinci Acetas
Zinci Chloridum

THE FEDERAL COMMUNICATION COMMISSION AND ITS PROPOSED ACTION ON DIATHERMY INTERFERENCE

The Federal Communication Commission is an attempt to prevent interference to F. M. and Television Broadcasting by diathermy or other electronic equipment of the non-radio category, is setting up certain standards of good engineering practice to be observed by manufacturers of this type equipment.

They intend to set certain frequencies of operation and tolerances for such equipment, and issue certificates to the manufacturer of the equipment, if their equipment passes certain required tests.

The requirements do not mean that the doctor has to discard his present equipment. However, he can satisfy himself as to what can be done to help him in the following manner.

Register his diathermy with some good service organization—preferably one which specializes in his type of equipment. The service shop will inform him as to practicability and cost of making the diathermy meet the specifications.

If the doctor purchases new equipment, obtain from the seller a money back warranty that the diathermy meets the F C C requirements and that the manufacturer is cooperating with the F C C in adapting good standards to such equipment.

As far as present equipment is concerned, it is not necessary to trade in or otherwise dispose of perfectly good equipment. It may happen that the diathermy meets all requirements, or is far enough away from Radio Equipment so that it does not cause interference.

References:

"Diathermy Standards"
Electronic Industries, July 1946, p. 151

Federal Communications Commission
Public Notice 84900, Sept. 25, 1945
also 71021 Oct. 5, 1943
also News Release 85742, Oct. 23, 1945

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BOOK REVIEWS

The Principal Nervous Pathways: By A. T. Rasmussen, Ph. D. New York, The Macmillan Company, 3rd ed., 1945. Pp. x + 73, figs. 28. Price, $3.50.

First published in 1932, this useful series of neurological charts and schemas with explanatory notes is widely and favorably known. It comprises twenty-eight plates (8 x 11 inches), chiefly representing functional pathways so drawn as to show both their anatomic relationships and the neuron chains composing them. Each plate is labeled and faced by a page or more of explanatory notes. The subjects of the plates are well chosen for their practical importance in medicine. In preparing the third edition the author has modified some of the charts of functional pathways in accord with recently established findings. The volume is recommended, for use either as by students as an atlas supplement to a text or as an independent reference for readers who are already familiar with the general organization of the nervous system.

HAROLD CUMMINS, Ph. D.

Howell's Textbook of Physiology: Ed. by John F. Fulton, M. D. Philadelphia, W. B. Saunders Company, 1946. Pp. 1304. Price, $8.00.

Although this textbook reports to be the fifteenth edition of Howell's Textbook of Physiology, it should be emphasized that it is virtually a new contribution ably edited by Dr. Fulton with the aid of some twenty-three contributors. It differs from the majority of the recent textbooks in physiology in minimizing clinical application and in presenting the material from the point of view that physiology is an academic discipline in its own right. This does not imply, of course, that the textbook will be of no interest to the clinician. On the contrary, the latter will find it an unusually well written and valuable account of the modern concepts of modern trends and beliefs in physiology.

The first section (about 40 per cent of the book) is devoted to a comprehensive and splendidly written section on the principles of nervous and muscular activity and on the nervous system, which leaves very little to be desired. The succeeding sections on blood and circulation are not quite as successful. The authors have experimented with a novel presentation of the material, but in doing so seem to have sacrificed unity and completeness. The problems of shock and hemorrhage, for example, would seem to warrant more specific and complete treatment than that contained in the few pages devoted to these topics. On the other hand, many of the individual chapters are to be recommended. The chapter on hemodynamics is particularly well done. The remainder of the book is taken up with discussions of respiration, body fluids and kidney, digestion, and metabolism and nutrition. The final section presents an excellent discussion of the physiology of reproduction.

Of interest and perhaps the only serious criticism of this otherwise fine contribution is the fact that the authors did not include a section dealing specifically with the endocrine glands. While there are references to the thyroid, adrenal, etc. in various discussions, there is no detailed information or emphasis as to the role of the endocrine glands as the second great coordinating system of the body. The parathyroid is not even mentioned in the index.

H. S. MAYERSON, Ph. D.

A Malariologist in Many Lands: By Marshall A. Barber, with a Foreword by Paul F. Russell, Lawrence, Kansas, Univ. of Kansas Press, 1946. Pp. 158. 7 pl. of photo. Price, $2.50.

This is a most entertaining and informative little book, of interest to malariologists, physicians and to laymen who are interested in the main highways and some of the by-ways of development of knowledge in the field of malaria, together with methods for its diagnosis and control.

Although Dr. Barber first became acquainted with malaria parasites of man a half century ago while a junior instructor at the University of Kansas (at a time when malaria was still present in that state), his intensive study of the parasite and the syndromes which it produces began in 1920 when he joined the U. S. Public Health Service. His intensive blood-film and spleen surveys, as well as anopheline-host transmission studies in the Southern United States were followed by similar undertakings in Tropical America, the Philippines, Malaya, the Fiji Islands, Equatorial Africa, Egypt, Macedonia, Cyprus, Turkey, the U.S.S.R. and India. In addition to accumulating a wealth of epidemiological data, Dr. Barber will be known for three particular contributions in malaria, namely (1) thick-blood films for diagnosis of the malaria parasites, (2) introduction of Paris green as a larvicidal agent for control of anopheline mosquitoes and (3) demonstration that plasmoquin as a therapeutic agent in human malaria sterilizes the circulating gametocytes, so that susceptible mosquitoes taking blood meals from human cases do not become infected. It may be of interest to add parenthetically that before 1920 Dr. Barber had developed the micro-manipulator for isolation of individual bacteria, and that he demonstrated this to the astonishment of the great Robert Koch.

The story of Dr. Barber's adventures in the field of malariology is told in his own inimitable way, as, for example, on page 50 his statement that "when the Devil sowed malaria, he took care to supply it with plentiful and efficient vectors." The reader's attention is maintained from beginning to end. Frequently in the midst of a running narrative one suddenly finds set down valuable diagnostic technics.

The print is clean and the little volume simply but attractively bound. The introduction is by Col. Paul F. Russell, M.C., A.U.S., who is a world authority on malaria.

"A Malariologist in Many Lands" is heartily recommended to the weary physician who desires a change from the usual type of detective story.

ERNEST CARROLL FAUST, M.D.

The Modern Attack on Tuberculosis. Revised Edition: By Henry D. Chadwick, M.D.,and Alton S. Pope, M.D., Rev. Ed. New York. The Commonwealth Fund. Price, $1.00.

This small manual opens with a quotation from an address delivered by Dr. Wade H. Frost in 1935, "The eradication of tuberculosis is now an expectation sufficiently well grounded to justify shaping our tuberculosis control program toward this definite end." It concludes with a forecast: "The bells that ring in the year 2000 may sound the death knell of the tubercle bacillus." These two optimistic predictions are well supported by the collection of data presented in the six chapters of this handbook on tuberculosis control. It was the aim of the authors not to present new but rather to collect for the tuberculosis work a mass of data ordinarily disseminated throughout the medical literature.

It is interesting to see that the emphasis is placed on the individual patient with tuberculosis not as a therapeutic problem, because adequate means of therapy are available, but as a focus for preventing perpetuation of the communicable disease. The publicized increase in morbidity rates from tuberculosis among the aged is believed to be only a transient phenomenon. The sanatorium as a means of controlling tuberculosis by isolating those most likely to transmit the disease is emphasized strongly. Now that photoflourographic methods are available for progressively increasing segments of the population, the authors urge intensive "follow-up" procedures so that the unsuspected case and the contacts of known cases may be repeatedly examined. Very simply they illustrate the necessity for this. In both instances, the unsuspected case and the contact, the chances are very great that the tuberculosis will be only minimal. By way of contrast, they cite the fact that even today about 25 per cent of all patients admitted to tuberculosis sanitoria have hopelessly advanced disease.

This book is recommended to any physician who wants to understand the methods by which public health authorities hope eventually to eradicate tuberculosis.

Sydney Jacobs, M.D.

Cornell Conferences on Therapy, Vol. I. Ed. by Harry Gold, M.D. New York. The MacMillan Company, 1946. Pp. 322. Price, $3.25.

Therapeutic conferences may be arranged in seminar style or in one where frank differences of opinion are encouraged. The latter was the one chosen when representatives of the various clinical and pre-clinical groups of Cornell University Medical School inaugurated their conferences for medical students in 1937. Because these became so popular and soon attracted not only students and residents but also many visiting physicians, it was decided to record stenotypically everything that was said at each conference. This material has been properly edited and presented as the first volume in what should be a most interesting series.

The diversity of subjects in the first volume compels attention. From such points of departure as the contents of a doctor's bag (many doctors' bags were examined, and most held little material for rendering first-aid to the more commonly encountered emergencies in general practice) to the Rh factor, the groups of consultants exchanged opinions. The clinically useful is constantly stressed.

Regardless of how limited or how broad his clinical interests, any practitioner can learn much from reading this delightful book. The exercises in clinical thinking contained are most stimulating.

Sydney Jacobs, M.D.

Theory of Life, Disease and Death: By Morton Whitby. Chicago. Cutler Publishing Co. 1945. Pp. 275. Price, $5.00.

The author, a member of the Royal College of Surgeons and a fellow of the Royal Society of Medicine, has concluded that life exists only because of the electrical energy which is present in the living substance. This is the mystic power controlling inorganic or organic matter and is the "symbol of God." Death is a loss of this electrical energy, while disease results in the transfer of energy to the area of disease to the detriment of the organism as a whole. Accordingly, the measurement of the electrical pattern becomes of paramount importance. If this is done, with care and patience, by means of the Ellis Micro-Dynameter (seven U. S. patents), one can make an accurate diagnosis of disease with the help of only a few biochemical analyses. The apparatus is also invaluable in the "dynamic treatment" of disease.

In these hectic days when life and the practice of medicine have become complicated almost to the breaking point, it is reassuring to discover at least one individual who feels that much of this effort is unnecessary, and that the struggle can be resolved so simply. In face of an acute paper shortage, however, the reviewer wonders how or why this book was ever published. It may be only coincidence that it was published in Chicago where the dynameter was designed.

H. S. Mayerson, Ph.D.

A Blind Hog's Acorns: Vignettes of the Maladies of Workers: By Carey P. McCord, M.D. (Illustrations by Francis Strobel). Chicago, Cloud, Inc., 1945. Pp. 311. Price, $2.50.

This book, a narrative autobiography of experiences in industrial hygiene, is true to the interesting type of story expected from Dr. McCord by those who know him personally or have heard him at the banquet table during meetings of industrial physicians.

The enthusiasm that Dr. McCord has put into a career of investigation and control of industrial disease registers as the experiences are recounted and the frequently ingenious solutions are given. This book will probably have its greatest appeal to those persons whose work takes them into the field of industrial hygiene. Any student of industrial hygiene would profit from these easily read accounts of industrial illnesses.

This reviewer feels that the choice of the artist who drew the illustrations might have been more happily made. Somehow the illustrations do not catch the tone of the narrative and the artist just cannot draw blind pigs.

Ralph H. Heeren, M.D.

New Drugs: By Arthur D. Herrick, with a foreword by Dr. Austin E. Smith. 319 pages, buckram. Published by Revere Publishing Company, 11 Stone Street, New York 4, N. Y. 1946. Price, $4.00

"New Drugs" consists of a description and discussion of the legal procedures necessary before a new drug can be made available for general use. As such, it is concerned chiefly with Section 505 of the Federal Food, Drug and Cosmetic Act and with regulations under that section of the Act. It may well serve as a guide regarding the requirements and policies of enforcement of the law and consequently should be of particular interest to drug manufacturers and distributors who are directly concerned with the introduction of new drugs. Since the book is written in the light of experience with existing laws it should also be a useful guide in the enactment of similar laws by states, municipalities or by other countries. Such questions are answered as: What constitutes a new drug? What drugs are exempt? What is the procedure necessary by the distributor for application for the release of a new drug? What type of evidence must be presented to establish its safety and how may such evidence be obtained? A number of appendices make up almost half of the volume. Appendix A is the "Report of the Secretary of Agriculture on Deaths Due to Elixir Sulfanilamide," which concerns a tragedy responsible in no small part for the enactment of legislation concerning the introduction of new drugs. Appendix B is the "Federal Food, Drug, and Cosmetic Act and Regulations." Appendix C records "State and City New Drug Legislation," while Appendix D is the "Official Rules of the Council on Pharmacy and Chemistry of the American Medical Association" which have been somewhat revised since the book was written. "New Drugs" concerns a field of endeavor not usually realized by the general public but established for its protection. Even a large part of the medical profession is unfamiliar with this phase of drug control or takes it for granted. Although the information is most valuable to the manufacturer and distributor of drugs it should also be of interest to the medical profession and even to the general public.

Ralph G. Smith, M.D.

Principles and Practice of Tropical Medicine: By L. Everard Napier, C.I.E., F.R.C.P. New York. The MacMillan Company. 1946. Pp. 917, Price, $11.00.

This new volume by Dr. Napier is a revision and an expansion of his earlier work which was reviewed in the New Orleans Medical and Surgical Journal, June 1944, p. 607. The volume now offers a complete picture, and a very satisfactory one, of tropical diseases. In the introduction, among many pertinent observations, the author warns against allowing the laboratory aspects of diagnostic methods to dominate one's thinking, which this reviewer considers most timely in the light of the mistakes he has seen made in the diagnosis of leprosy by too slavish adherence to the results of laboratory tests. The new parts of the book, making up about one-third of the total of 784 pages, covers treponema infections, skin diseases, the predominantly tropical venereal diseases, nutritional diseases and parasitic diseases. As the review referred to above covered sufficiently the contents of the earlier volume these comments will be confined to the newer parts of the work. The controversial question as to the relation of yaws and syphilis, the author resolves by regarding the causative organisms as "closely related but of quite distinct species." The author considers that both diseases "arose from a common ancestor, probably in prehistoric times." In the parallel column differentiation of the two diseases, the writer places the response to mercurial treatment as one of the features; since mercury is now rarely used in syphilis this probably is not very helpful. Penicillin is mentioned in the treatment of yaws, illustrating the pains the writer has taken to bring the book up to date. Under the discussion of prevention of tropical ulcer the writer points out the fallacies that are inherent to generalizations in fields where our knowledge is limited. To one interested especially in preventive medicine it is discouraging, but not surprising, to find that the author expresses the opinion that ringworm of the feet is almost a concomitant of living in the tropics. The sections dealing with medical parasitology consider the diseases primarily from the point of view of the clinician but public health and epidemiological aspects are particularly competently handled. The section on nutrition and nutritional diseases in the tropics starts with general consideration of diet with special attention given to rice which is so largely eaten in warm climates. The economic factors are given adequate attention. While the author gives full credit to Goldberger for his monumental work in pellagra he feels "that no explanation of the etiology of pellagra can be accepted that does not take into consideration the past and present predominance of the disease among people whose staple food is maize." In the section on rabies there is a very good appraisal of the circumstances that should lead one to administer antirabic vaccine. The book as a whole can be recommended without reservation to students and physicians who need to learn about and to deal with diseases occurring predominantly in warm climates.

G. W. McCoy, M.D.

Proceedings of the Rudolph Virchow Medical Society. Vol. 3, 1944. New York, Brooklyn Medical Press, 1945. Pp. 104. Price, $2.00.

A volume of interesting articles on subjects, medical, surgical, roentgenologic and the specialties. The articles are brief and interesting. The size of the magazine does not justify detailed description of the contests, but anyone will find an hour of pleasant and profitable reading.

I. L. Robbins, M.D.

PUBLICATIONS RECEIVED

Commonwealth Fund, New York: Psychotherapy in General Medicine, by Geddes Smith. Medical Education and the Changing Order, by Raymond B. Allen, M.D., Ph.D.

Lea & Febiger, Philadelphia: Urologic Roentgenology, by Miley B. Wesson, M.D.

New Orleans Medical
and
Surgical Journal

Vol. 99 OCTOBER, 1946 No. 4

SOCIALIZED MEDICINE[*] THE PHANTOM APPROACHES

HOWARD MAHORNER, M.D.
NEW ORLEANS

The term socialized medicine is not intended to designate companionable or friendly or humanitarian medicine. Socialized medicine denotes the plan of medical care consistent with a political viewpoint of socialism which may be defined as "a political and economic theory of social reorganization, the essential feature of which is governmental control of economic activities to the end that competition shall give way to cooperation and that the opportunities of life and the rewards of labor shall be equitably apportioned."

No doctor who has had the responsibility of life in his care and who repeatedly has had to make decisions or perform technical procedures upon which life and death may be determined, could do other than approve the idea that all humanity—each man, woman and child—deserves the most that medical science can offer to ameliorate the suffering of their illnesses and prolong their life in health. Unfortunately the best of medical science is not available to every individual of the world. Let it be acknowledged with little for controversy that as a people or a nation, the people of the United States have the best medical care in the entire world. This is no vain boast. It has been recognized as a fact by doctors for generations that the facilities and abilities in the medical centers of the United States have been superior to those of other nations.

Unfortunately, however, even in the United States satisfactory medical services are not always available to everyone. The reasons for this are the changing type of practice, the increased cost, the lack of medical facilities and absence of doctors in certain sections. Formerly the family doctor performed most if not all of the professional needs for a family. He delivered the children, directed their care and medicines, fixed fractures and did an occasional appendectomy. But today, especially in medical centers, he is not as often found. Why? Because the tremendous expansion of knowledge has made it impossible for anyone to be thoroughly versed in each of these lines and because specialists with a high degree of information and skill in different branches are sought, wherever available, when a difficult clinical problem arises.

The family doctor is often praised for his diagnostic acumen, his skillful insight and expert senses, but in reality, they were not more keen nor more accurate in physical diagnosis without laboratory aids than is the doctor of today. The doctors of previous generations were often incorrect, but this was not proved to them by methods of precision in diagnosis, such as exist today. That is not to disparage the value or the efficiency of the doctors of past generations; for, lest the physician or surgeon of today become too proud of his comparative knowledge and skill, let him admit that fifty years hence he will appear poor indeed when compared with the skill and exactness which will have developed since now. Precision instruments and facilities are most expensive, an economic factor with which the older family doctor did not have to contend; but the modern doctor knows he must have them and use them.

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[*] Delivered at a joint meeting of the Women's Auxillary of the Mobile County Medical Society and the Mobile Civic Clubs, Mobile, Alabama, March 29, 1946.

Besides the changes in the type of medical practice, numerous other factors combine to make the cost of medical diagnosis and care much more dear than formerly. These factors include expensive laboratory tests, instruments, expensive drugs, complicated administration of drugs and special nursing care for sick patients. In addition to this the rapid expanse of knowledge necessitates continued research and continued re-education of physicians by travel and attendance at meetings.

Hospitalization is much more often essential now than formerly, because so much more can be done to cure patients promptly and completely. Roentgenograms (x-ray examinations) are much more frequently needed now than formerly because certain lesions can be diagnosed only with the aid of x-ray. For example, cancer of the stomach in a stage when it can be removed by an operation may be diagnosed accurately only by the x-ray. All of these factors have tremendously increased the cost of diagnosis and treatment; all of them have tended to make modern medicine unavailable to more and more people.

The reasons are not economic alone, they are geophysic as well. There are more doctors per capita in metropolitan areas, fewer in rural areas. Wealth is not the only factor influencing this. Other causes for this unequal distribution are the availability of medical facilities and the presence of other experts. Some of the highest per capita doctor to patient ratios in the country are in small towns under 35,000 where the profession consists of experts with precision facilities.

The cry on the part of politicians that doctors are not well distributed is in reality more a problem of transportation than of the existence of medical facilities. Able doctors demand and help to create the necessary facilities including laboratories and hospitals. A state is far better off to have a few centers with experts and expert facilities and a scarcity of doctors in its extremely small communities than to have an abundance of widely disseminated doctors without medical centers. People may recover from mild illnesses. They must have experts with proper equipment to pull them through some of the serious illnesses. Somehow all of this and in the best of form, should be made available to all people.

The method of accomplishing this has been the thought of many men; political economists, politicians, statesmen, organized medicine, the doctors and the people themselves. The proposals of designs for its accomplishment vary from pure communism and socialism to more conservative changes; and admittedly in some small sources there is pure obstruction to any change.

Various health bills and proposals have been introduced in numerous states and in the Congress of the United States. Among the most important are the Hill-Burton Bill (S-191), the Wagner-Murray-Dingell Bills, and the National Health Message of Harry S. Truman, President of the United States.

The Hill-Burton Bill (S-191) was introduced into Congress January 1945, by Senator Hill (Alabama) and Senator Burton (Ohio). It has been passed by the Senate. It provides for survey of public health facilities and for construction of hospitals and public health centers. It authorizes an appropriation of $75,000,000.00 annually for each of five fiscal years for the construction program. Other than minimum requirement standards, it divests the federal government of its control and leaves the operation and management of the institutions to local and state agencies. Such a program the people, doctors and organized medicine can approve and applaud. The first step in providing adequate medical care for people is to furnish the facilities. This plan has the laudible provision, in the version of the bill passed by the Senate, that the federal government does not seek control of either the patient or the doctor. This is legislation we should all approve.

The Wagner - Murray - Dingell Bills (S-1050 and S-1606), were introduced in the Senate by Senator Wagner of New York and Senator Murray of Montana while companion bills (H.R.-3293 and H.R.-4730) were introduced in the House of Representatives by Congressman John Dingell of Michigan. This is an omnibus bill comprising social security compulsory health insurance, sickness benefits, public health works. It will provide medical care for 120 million of our 135 million people. It gives absolute powers of control to the Surgeon General of the Public Health Service under direction of the Federal Security Administrator. It will control patient and doctor alike, and its provisions are at the discretion of the Surgeon General. It provides for grants and loans for hospitals and public health centers, for maternal and child welfare services. It provides grants to states for public assistance for needy individuals including medical care for such individuals.

This type of medical care is not free. The cost comes in the form of increased taxes. The cost is an 8 per cent tax on income, — 4 per cent on employer and 4 per cent on employee, — on wages up to $3,600.00 annually. The self employed would be taxed 5 per cent. The total would be an annual tax of about four billion dollars. The operation of the plan is to be centrally controlled in Washington. It would furnish, after its own fashion, medical services for 120 million people of the United States, and though the doctors would not be in uniform, they would be so regimented, by necessity of complying with the politically controlled form of medicine that it would, in reality, be true socialization of medicine. A bureau with approximately 300,000 employees would be the result of this plan. Regardless of what is said to soften the idea, the doctor and the patient would be controlled in many respects by the government, because the government would pay the bills, and no provision is made in S-1606 for divesting the central government of its control and putting the control in the hands of local agents, similar to that provided for in the Hill-Burton Bill. This is socialized medicine. It is Russianized medicine or Nazified medicine, both of which have been shown to be less efficient than even the open plan operating in the United States today.

Attempts at government control of medicine are very old, dating back in some forms for centuries. The Germans and Russians had socialized medicine before World War II. Notably the quality and quantity of their medical service was inferior to ours. Medicine in a state like Germany and Russia *must* be socialized. They operated on a different political theory, which we Americans generally do not like.

President Truman's National Health Program was read to Congress November 19, 1945. In it he stated, "I repeat what I am recommending is not socialized medicine. Socialized medicine means that all doctors work as employees of government. The American people want no such system. Under the plan I suggest our people would continue to get medical and hospital services just as they do now, — on the basis of their own voluntary decisions and choices." Under the Wagner-Murray-Dingell Bill all doctors or practically all doctors will work for the government controlled system — in essence they will work for the government and President Truman admits that the American people do not want that type of medical service. Again he said, "Medical services are personal. Therefore, the nationwide system must be highly decentralized in administration. The local administrative unit must be the keynote of the system." The Wagner-Murray-Dingell Bill provides for centralization so strong it could not fulfill the requirements of the President's National Health Program.

Wendel Berge, Assistant Attorney General of the United States, in an address advocating an expansion of medical services in the form of a national health program said, "Support of the doctors is essential to the salvation of the movement. If doctors oppose or stand on the sidelines, the laymen will create a medical order which may prove to be indifferent or even blind to the values doctors prize most." Let it be known now that doctors individually and collectively oppose the creation of any such order as that advocated in the Wagner-Murray-Dingell Bill.

If not the Wagner-Murray-Dingell Bill, what then to ease the individual financial burden of sickness and bring better health facilities to more people?

The following expansion would seem a step in the right direction and would not create political medicine nor advance into the design of socialism or communism.

1. Construction of adequate facilities by measures such as those embodied in the Hill-Burton Bill.
2. Provision of transportation to bring the seriously sick to medical centers; centers where and only where, the more complicated diseases may be treated with the most modern methods.
3. An extended program for disease prevention with the development and extension of public health service.
4. The development and extension in all localities of voluntary or community prepaid sickness insurance plan.
5. Providing of medical facilities and other attractive inducements by smaller communities to stimulate the dissemination of more doctors in the rural areas.
6. The provision of hospitalization and medical care to the indigent by local authorities.
7. The system should not be under totalitarian control of Washington.

Civilian steps have already been started in these directions. One of the best and more magnanimous is the Blue Cross Plan which provides hospital care for a nominal fee to over 22 million Americans (16% of the population). The cost is fifty cents a month for an individual. The organization is non-profit and its operation requires very little overhead. Those insured in the Blue Cross Plan are entitled to thirty days' hospital care, operating room services, routine laboratory service; general nursing care, and a discount for 50 per cent on x-ray tests. The best doctors in the world cannot properly handle difficult problems without good facilities. There are many other factors in providing good medical care but the basis of it is modern facilities. The Blue Cross Plan could be extended to include everyone. A collateral expansion of this non-profit community sponsored Blue Cross Plan is one providing for surgical care. The cost is 75 cents a month and it provides maternity care and surgeon's fees according to a scale. In New Orleans beneficiaries have found that 97 per cent of the surgeons' fees are entirely covered by the plan. In Alabama a similar plan is in operation to provide surgical and medical fees.

Let us not regiment our doctors and patients, by giving the federal government control and say so of this part of their lives. Security or the false promise of it was made in Germany. They had socialized medicine. Security is not to be purchased by freedom. Government control of medical profession and patients is legal under such measures as the Wagner-Murray-Dingell Bill provides. The promise of security it offers is tempting; but when it has to be purchased with liberty as well as with tremendously increased taxes it becomes too dear and even abhorrent.

One would think that health is an individual and a national thing, that it is the province of government to see that each one is kept well for the good of the whole. What about bread and food? Proper nourishment of the individual also is essential for the good of the whole. If the federal government takes over the duty of providing medical care for the people, perhaps it should go back and provide food for all people by taxing the entire population and distributing the food. And what about justice? Certainly that is a basic right of each individual. Why should not the government take over the legal profession, tax the people, and pay each attorney what is just for each of his cases? These thoughts are all analogous; they are un-American and it would appear that once initiated by law they would be an irrevocable step towards regimentation of the people in national totalitarianism.

Doctors know some changes are necessary in the program of medical service. These are necessary not in spite of them, but because of them. Doctors, and the advances in scientific medicine are responsible for all this change in medical care. They will welcome and help with the changes necessary to take good medicine to the people. Let us make these changes the American way from the people and by the people. Let us not make the medical profession a federal or political agency. By doing it the American way we will have better medical care and a medical order more vast and mightier than anything we know. Socialization and central control of medicine on the other hand will result in a deteriorization of medical service — directives, inefficiencies, requisitions and inevitably more unhappiness for doctors and patients. Let us defeat the Wagner-Murray-Dingell Bill and let each community's medical profession and citizens work to establish some local non-profit pre-payment insurance plan for sickness.

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FIVE YEARS' EXPERIENCE WITH ELECTRIC SLEEP (ELECTRIC SHOCK) TREATMENT*

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NEW ORLEANS

In the past few years there has been a marked increase in interest in the new technics used in the treatment of mental and emotional disorders. Analysis will show that mental disorders in the military service in World War II have been better treated than they were 25 years ago, but the problem of the sick and maimed veteran is going to be a major consideration for the next fifty years. That emotional disorders in the general population have greatly increased during the war years and since the cessation of hostilities in Europe and Japan is an established fact; however, the more malignant psychosis, such as dementia precox, has not shown much if any increase.

Fortunately, today we have better facilities than ever before for treating those mentally or emotionally ill. The greatest therapeutic achievement has been made in all forms of emotional disorders. You will recall that insulin treatment was introduced and extensively used eight or ten years ago, and it actually is of great value in some instances, particularly in schizophrenia. Following the wave of popularity enjoyed by insulin, the drug metrazol was introduced, but there are many drawbacks to this form of shock treatment, and though it accomplished much in a therapeutic way it has been almost entirely discontinued.

Electric sleep (electric shock) therapy has so many advantages over insulin and metrazol that it has almost completely replaced these forms of treatment .Electric sleep treatment is now being used in hospitals for mental patients throughout the world.

While electric sleep therapy is used to treat dementia precox and hysteria, it is of paramount value in involutional melancholia, reactive depressions, the depressions which are part of the manic depressive syndrome, and other emotional disorders. It is used in treatment of the excited phase of manic depressive syndrome but the results are not so brilliant.

Those patients who are slightly or moderately depressed do not come to you asserting they feel blue, melancholic, or downhearted; rather, they have complaints referring to various bodily functions, such as weakness, being easily fatigued, loss of appetite, anorexia, loss of weight, insomnia, peculiar feelings in the head, eye symptoms, tachycardia, palpitation of the heart, sex disturbance (especially loss of libido), and so on. The examining physican can usually get at the true state of affairs if he will ask the patient the direct questions: "Are you happy?" "Are you sad?" "What gives you pleasure?" When these questions are put to a melancholic patient he generally stops talking about his heart or stomach, and tells about his depression. Frequently the patient has had one or more similar attacks previously.

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There are several well recognized types of depression or mental disorders in which the mood disturbance is of primary importance. They are:

(1) *Reactive depression, prolonged grief*: Here the patient is unable to recover from a bereavement or some other emotionally depressive experience, whereas on the other hand the average person would feel grieved but would soon be able to adjust and carry on. The patients we are discussing would for months or years continue to be deeply grieved and depressed and might cry most of the time. These patients respond brilliantly to electric sleep treatment, and recovery is almost 100 per cent.

(2) *The depressed phase of the manic depressive syndrome*: This represents a large percentage of people who are emotionally disturbed. There is often a history of previous attacks and more or less psychopathology in the family. This psychosis is looked upon as being due to some conditioning factor in the constitution of the patient, due in large part at least to hereditary influences. Electric sleep treatment restores these patients within a few weeks, thus saving the patient months of suffering while waiting for recovery which may take as long as one, two, or ten years. Suicide in these patients is always a real danger, and any shortening of the period of depression will lessen the risk of self-destruction.

(3) *Involutional melancholia*: This condition is frequently seen in women between 40 and 55 years of age, and in men between 55 and 65; however, it may occur later. Marked depression with agitation and self-accusatory delusions are present, and the patients are particularly apt to commit suicide. As a group, they respond well to electric therapy, recovery taking place in 70 to 90 per cent.

(4) *Presenile depression*: This usually occurs later than involutional melancholia and care must be taken to differentiate these people from those suffering with senile deterioration. Here response to electric sleep treatment is better than 70 per cent.

(5) *Psychoneuroses*: These as a rule do not respond well to electric sleep treatment unless depression is a major component in the disturbance. The obsessive compulsions and the tics do not do particularly well, though occasionally a person with hysteria will make a quick recovery after one or more treatments.

(6) *Depression associated with other diseases (as general paresis, tuberculosis, primary anemia, cardiovascular disease)*: These are well suited for electric sleep therapy, and brilliant results are often attained by helping these people over their depression. They are then able to cooperate in treatment of the primary disease.

While insulin, metrazol, and electric shock treatment were first used almost exclusively in dementia precox or schizophrenia, these treatments are tremendously more beneficial in the emotional disorders. We are still employing electric shock treatment and insulin in the treatment of dementia precox, and in early cases the results are better than by any other form of therapy. When the electric treatment does not give good results, a course of insulin treatment may be successful in greatly improving the patient so he is able to leave the hospital and return to his home. If he has a recurrence in a few years, he will probably respond satisfactorily to a second or third treatment.

The use of metrazol has been almost discontinued, though some hospitals still employ it in certain cases. I believe we have not given metrazol in New Orleans for the past five years though we still use insulin in a restricted number of patients.

Contraindications to electric sleep therapy are few. Marked osteoporosis, malignancy, cardiovascular disease with decompensation, acute febrile reactions, are absolute contraindications; but cardiovascular disease where compensation is adequate, a not too recent coronary thrombosis, chronic tuberculosis, arthritis, organic brain disease, are not contraindications to this treatment, though undoubtedly they add to the risk. Patients with hypertensive cardiovascular disease, even where the systolic blood pressure is over 250, usually do very well under electric sleep treatment, for the hypertension is often the result of agitation and mental turmoil; as the patient improves, the pressure lowers and often becomes normal for the patient's age.

Chronic tuberculosis or old established tuberculosis with tubercular organisms in the sputum can be successfully treated when the emotional disturbance requires therapy. Curare is used in some instances to "soften" the muscular contractions but it seems to add some further risk.

Electric sleep treatment is not in itself dangerous, but when used in certain conditions there is some danger; but there is danger in all surgical procedures, and in fact, our every day life is beset with danger. I frequently tell a patient this treatment is as dangerous as riding 10 or 20 miles in an automobile, crossing a busy city street, or having two or three teeth extracted.

We have treated over 3,070 people with electric shock therapy and have given 27,231 treatments without a fatality. There actually have been considerably more patients treated with this therapy than I have recorded, especially at the Charity Hospital and the City Hospital for Mental Diseases, but reliable records from those hospitals were not available.

Complications do occasionally occur, the most common being dislocation of the jaw. This is usually reduced while the patient is relaxed by treatment; sometimes we have given a patient with a dislocated jaw a second treatment and reduced the dislocation as soon as he relaxed. Fractures of the body of the vertebrae occur, but these are not the serious injuries we associate with traumatic jack-knife fracture (fracture-dislocation of the spine), for here the spinal cord is almost always injured, whereas if there is a fracture of the body of the vertebra under electric sleep treatment the cord is not injured. There may be some pain on movement and on deep inspiration for several days, but it is not necessary to put the patient in a cast or institute bed rest for a protracted period; these patients do very well under simple treatment. The most disadvantageous result is that treatment for the time being at least must be discontinued; however, after a week or two it may be commenced again, under the quieting effect of curare.

In the large series of patients that we have treated, there have been a few fractures of the neck of the humerus, not more than two or three, and one of the neck of the femur, due to sudden muscle contraction. These have occurred in elderly patients who might have received a similar injury by slipping on the floor in their own home.

In all the patients we have treated in New Orleans, we have not had a fatality, and throughout the world, where several million treatments have been given, less than a dozen fatalities have been recorded. All these were people who had some complicating disorder, as brain tumor, syphilitic meningitis, advanced age, or hypertension —not one of the deaths occurred where treatment alone could be held responsible. There have been so few deaths that postmortem studies are entirely inadequate, and there has not been a postmortem on an individual suitable for study (one between 20 and 40 years of age, free of any other disease). We do not hesitate to treat elderly people, and have had to treat a number over 70 years of age, but we do not like to treat individuals of this age any more than you would like to perform an operation on the same person; however, these individuals are sick, and if anything is to be done for them the added risk must be accepted.

TABLE 1
ELECTRO-SLEEP THERAPY, DEPAUL SANITARIUM

Year	No. of Patients	No. of Treatments
1941	153	1,436
1942	217	1,963
1943	121	2,658
1944	501	4,937
1945	547	6,170
1946 (Jan. to Apr.)	171	1,799
	1,716	18,963

TABLE 2
ELECTRO-SLEEP THERAPY, TOURO INFIRMARY

Year	No. of Patients	No. of Treatments
1941 (11/41-12/41)	45	85
1942	192	878
1943	265	1,521
1944	302	1,750
1945	340	2,084
Clinic	50	350
	*1,194	6,668

* Approximately 90 per cent were "out patients."

TABLE 3
ELECTRO-SLEEP THERAPY, TOTALS

Hospital	No. of Patients	No. of Treatments
DePaul	1,716	18,963
Touro	1,194	6,668
Baptist (approx.)	160	1,600
	3,070	27,231

Undetermined number of treatments at City Hospital for Mental Diseases and Neuropsychiatric ward at Charity Hospital.

Tables 1 and 2 show the number of patients at DePaul Sanitarium and Touro Infirmary that have received electric sleep therapy and the number of treatments given. Table 3 shows the total number of patients and the total number of treatments.

DePaul Sanitarium is the only large private hospital in Louisiana for neuropsychiatric patients, the capacity of which is about 225. The "open" staff of the Sanitarium consists of a medical director and psychiatrists of New Orleans, and there are consultants in all other medical specialties. Statistics in this paper represent activities of the staff of the Sanitarium; opinions and conclusions are those of the writer.

TABLE 4
DEPAUL SANITARIUM, 1945

Admissions	713
Discharges	709
Condition On Discharge:	
Restored or much improved	621
Slightly improved	44
Unimproved	20
Expired	14

No deaths as a result of electric sleep treatment.

Table 4 shows the activity of the DePaul Sanitarium for 1945.

It is generally conceded that electric sleep treatment is particularly well suited for treatment of depressive states, though for the first few years after their introduction the various shock treatments were used almost exclusively in the treatment of schizophrenia. The results obtained in the treatment of this disease are better than by any other therapy, but on the whole, the prognosis in schizophrenia does not permit one to be too optimistic. On the other hand, the depressions respond brilliantly, and we can promise nine out of ten depressed people that they can be restored in three to four weeks; usually six to ten treatments are all that will be required.

TABLE 5
MANIC DEPRESSIVE PSYCHOSIS (189)

	Age Groups						
	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Manic	4	19	14	12	9	3	0
Depressed	4	14	20	27	29	9	3
Mixed	2	1	8	5	2	1	0
Circular	0	0	0	0	1	0	0
Totals	10	34	42	44	41	13	3

Table 5 shows the analysis as to age and type of disorder in the 189 patients suffering from manic depressiive psychosis.

TABLE 6
MANIC DEPRESSIVE PSYCHOSIS

Condition on Discharge	Restored or much Improved	Slightly Improved	Unimproved
Manic	60	0	1
Depressed	160	0	0
Mixed	18	0	0
Circular	1	0	0
Totals	187	1	1

Table 6 further breaks down this group. It is seen that of 189 patients in this group 187 were restored or much improved. These figures are actually too high, but they represent the conditions of the patients when discharged from the DePaul Sanitarium. The follow-up study which is to be undertaken will certainly show that the very high recovery rate was to some extent unwarranted.

TABLE 7
INVOLUTIONAL MELANCHOLIA (25)

Age Groups:	40-50	50-60	60-70
	8	4	13
Marital Status:	Married	Single	Widowed
	15	2	8

Sex: Male	Female	Condition on discharge:	Much Improved or Restored	Unimproved
5	20		25	0

Table 7 shows the results attained in 25 patients suffering from melancholia. Here again the condition on discharge may not have been a true picture, but granting a certain percentage of over-optimism the results as we saw them were very dramatic and heartening.

TABLE 8
DEMENTIA PRECOX (103)

Age Groups	10-20	20-30	30-40	40-50	50-60
Paranoid	1	21	15	11	4
Catatonic	1	10	3	0	0
Hebephrenic	1	7	6	6	0
Simple	3	8	6	0	0
	—	—	—	—	—
Totals	6	46	30	17	4

TABLE 9
DEMENTIA PRECOX

Condition on Discharge:	Restored or much Improved	Slightly Improved	Unimproved
Paranoid	39	8	3
Catatonic	12	1	1
Hebrphrenic	15	5	0
Simple	16	3	0
	—	—	—
Totals	82	17	4

Tables 8 and 9 shows the results obtained in 103 patients treated at DePaul Sanitarium during 1945.

We treated 14 patients in whom alcohol or drug addiction was the principal or contributing problem in the illness. Thirteen were apparently restored or much improved. A follow-up will throw some light on this group.

Certain selected patients suffering from marked psychoneurosis (32 individuals) were treated, and most of them greatly improved.

Electric sleep treatment has proved of great value when used "symptomatically." There are a certain group of patients, mostly schizophrenic, who can be greatly improved physically and mentally by an occasional series of electric sleep treatments. We give one treatment a week to an old lady who was a patient in a state hospital for five or six years; now, with the one treatment a week, it is possible for her to live with her son who is a physician in New Orleans and she is able to care for her grandchildren. When the treatments are discontinued, she soon becomes confused and deeply depressed and is then unable to do anything for herself or anyone else. For approximately three years she has, by the use of electric sleep treatments enjoyed a relatively normal life. Great benefit in other chronic patients has been brought about by the occasional use of this type therapy, and less restraint or sedation has been required.

CONCLUSION

(1) Electric sleep (electric shock) treatment is safe, particularly with patients who might be considered good surgical risks.

(2) Almost 28,000 treatments were given 3,070 patients, with no fatalities and relatively few dislocations or fractures, less than 10 per cent.

(3) All depressions should receive this form of therapy even when the psychosis is of several years' duration.

(4) In schizophrenia, better results are obtained through electric sleep treatment than by any other method; and the shorter the duration of the illness, the better the results. Insulin is superior to electric sleep treatment in certain cases of schizophrenia.

(5) Electric sleep (electric shock) treatment is valuable in maintaining certain chronic cases at a higher social adjustment than they could otherwise obtain.

I wish to express my appreciation to the Administration and Staff of the DePaul Sanitarium for their help and cooperation; particularly am I indebted to Miss Elizabeth Haik, Librarian, for the research and study that she carried out.

DISCUSSION

Dr. Walter J. Otis (New Orleans): Brain stimulation and not brain destruction should be the aim in the therapy of most mental disorders.

It may seem strange that after spending many years in trying to stop convulsions we now help to produce a convulsive reaction.

Dr. Holbrook has brought to your attention the results of this particular type of therapy over a period of years, covering a number of treatments given to a number of patients, material having been secured from various sources with the kind permission of the neuropsychiatric group here in New Orleans. The inception of the electric sleep therapy was inaugurated in Louisiana at De Paul Sanitarium in January, 1941. The results have been most satisfactory and we are hopeful of a continuance in further cases presenting themselves for treatment. As has been told you the earlier they are presented to the neuropsychiatrist the better the prognosis. It is hoped that the physicians from a medical standpoint detect earlier any prodromal symptoms or syndrome which would lead them to believe that the patient is becoming a neurotic, psychotic or psychoneurotic, thereby availing themselves of one of the newer therapies being used with success.

Dr. J. P. Sanders (Shreveport): All of us have realized in the institutions with schizophrenics who have dropped down as low as human being can if you can raise their status to where they can feed themselves and bathe and go to the table and eat, it is a great help. There isn't any question about it, this is an enormous problem. If you can do that much for those it is a great help. Maybe as time goes on and we can have more work done in our grade schools and grammar schools in educating introverts, potential schizophrenics, maybe we will not have to enlarge our insane asylums.

Dr. R. H. Foster (Pineville): I would like to confirm what Dr. Otis and Dr. Holbrook have said about the benefit to the schizophrenic patient in State Hospitals. At the Central Louisiana Hospital we have been using this treatment for four and a half years. A number of schizophrenic patients responded quite well. The various types of depression nearly always recover from shock treatment, and are able to leave the hospital. The first case treated showed a surprising recovery and improvement. She was a catatonic praecox, who had not talked, nor eaten by herself for more than four months. Soon after treatment was started she began to talk and eat and rapidly recovered and was paroled. Later she had a relapse and shock treatment this time did not help her. She was put on sub insulin shock treatment and recovered again and has now been out of the Hospital about three years. Another advantage of shock treatment, as has been brought out, is the applicability in ambulatory cases. There is no doubt that early treatment is most valuable, and this is where we are handicapped in the State Hospitals as we do not get these patients until everything else has been tried, and many times the true mental condition has not been recognized.

In the involutional cases, prior to electric shock, we relied on stilbestrol, but it was not very efficacious. With electric shock treatment practically all of these patients recover.

Dr. N. F. Thiberge (New Orleans): I should appreciate it if Dr. Holbrook would give us in some detail how treatment is applied. Most of us are not familiar with the method used.

Dr. Joe Holoubek (Shreveport): In cases of alcohloics with major depressive states, I wonder if you will compare the electric shock treatment with the ordinary psychiatric treatment of alcoholics. We know that many of these patients are emotionally immature and I wonder if this treatment is better than psychotherapy or psychoanalysis in the chronic alcoholic.

Dr. Charles S. Holbrook (in closing): For many years it was demonstrated that small amounts of electricity applied to the brain surface produced certain responses to those motor areas which could readily be defined. If a larger amount of electricity were used, the animal tested might have a convulsion. No clinical use was made of this application of electricity until quite recently. For eight or ten years insulin and metrazol were used to treat patients with mental disorders, and the principal immediate effect of the use of these drugs was the induction of a convulsion, and especially was this true of metrazol.

Treatment with insulin and metrazol had many objections and considerable risk for the patient could not be escaped. Electricity was then employed to produce the same results that were obtained from the employment of the two drugs mentioned. This method has come into general use in all our well regulated psychiatric hospitals. Seventy to 150 volts are applied to the temples for 1/10, 1/20, or 1/2 second, producing immediate loss of consciousness and a convulsion which lasts 35 to 45 seconds. The patient often goes into a deep sleep and may be confused and restless for a time. In experimental animals the safety margin is about 500.

Many patients are treated as "out patients." They report to one of the hospitals about eight o'clock in the morning and are back home by nine-thirty or ten o'clock. Some men even go to their work in the afternoon, though this is not recommended.

Patients do not dread the treatments, and if they should begin to feel another period of depression developing, they come and ask to be given more treatments.

While we have given over 28,000 treatments, we have never had a death resulting from this therapy. There have, however, throughout the world, been some 12 or 13 deaths, but these occurred in people who had something else (brain tumor, high blood pressure, advanced senile changes, or some other serious complication) wrong with them. Postmortems have been extremely few and no suitable studies have been made because death has not occurred in a patient without complications. The most suitable type of patient would be one between 20 and 35 years of age, in good health, and free of complications. No patient like this has died as a result of electric sleep (shock) therapy.

I usually tell the families of my patients that there is some risk to this treatment, as there is to almost anything, but the danger is no greater than driving ten miles in an automobile, crossing a busy street, or having a couple of teeth extracted.

When it comes to alcoholics, we are not so sure of our ground. Insulin was first introduced into psychiatry because it was used in the treatment of alcoholism, and it was observed that after these individuals were treated they experienced some change in personality and often seemed much improved. Shock was then used in the treatment of dementia precox, and later in treating patients with depression. In the latter group we have achieved brilliant results. While electric sleep (shock) is sometimes used in alcoholism, we do not consider it the treatment of choice—I prefer to use "condition reflex treatment" and if patients wish to be helped, a great deal can be done for them by this form of therapy.

Electric sleep (shock) treatment is especially beneficial in the treatment of all kinds of depression, and it is really a godsend to these patients, as they frequently recover in a few weeks from a disorder which might otherwise run many months or even years and might drive the patient to suicide.

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THE USE OF BIOPSIES IN THE DIAGNOSIS OF CANCER*

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One of the most encouraging developments in cancer control during the past twenty years has been the steady and rapid improvement in the efficacy of treatment. Most patients with cancer can now be offered an encouraging prognosis if the disease is discovered and treated in its early stages. Modern surgical and radiologic methods can effect a five-year cure in more than 70 per cent of the early cases of carcinoma of the skin, lip, breast and cervix and in a very good proportion of other malignant lesions. If cancer is neglected in its initial stages and allowed to progress untreated the patient's prognosis rapidly deteriorates. Once metastases have taken place the chances of cure become remote. Warren and Hoerr,[1] in a study of over 500 cases of carcinoma of the skin, showed that the size of the lesion at the time of treatment was more important than any other factor in determining the prognosis. In patients with lesions measuring less than one centimeter in diameter the mortality rate from cancer was only 7 per cent as contrasted with a mortality rate of 82 per cent in patients with later lesions measuring five centimeters or more in diameter. In this country approximately one of every ten deaths is a direct result of malignant disease. Without any improvement in present methods of therapy this toll could be cut in half by early diagnosis and prompt, adequate treatment.

One important factor which contributes to the delay in the diagnosis of cancer is that the early lesions are often insignificant in appearance and seldom cause distressing symptoms. Patients often postpone a visit to the physician until the disease has progressed beyond the curable stage. The present campaign in Louisiana and other states to reduce the death rate from cancer is being directed almost entirely toward the general public. People are being urged to seek medical advice at the first appearance of any abnormal tissue mass, any unexplained bleeding or any ulcer which does not heal in a reasonable time. If this campaign is effective, physicians may expect to see more patients with minor lesions of questionable nature and will face the responsibility of making a prompt decision as to whether each lesion is benign or malignant. Clinical judgment may not be adequate to this task but pathologic examination of a representative piece of tissue will usually reveal the nature of a malignant growth while it is still small, localized and curable. Improvements in the surgical and radiological treatment of cancer have further increased the need for pathologic diagnosis. It is not enough to know whether a growth is benign or malignant; the choice of treatment may depend on the type of neoplasm and to a certain extent on the histologic grade of malignancy. Pathologic diagnosis is so firmly established as an essential part of the proper management of suspected malignant disease that there is no need to discuss its advantages. There are, however, a number of disadvantages and shortcomings which serve to discourage some physicians from making full use of this important diagnostic aid.

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One of the most serious disadvantages of biopsy is its cost. Reliable pathologic diagnosis requires the services of a skillful physician to remove the tissue, a good technician to prepare the slides and a competent pathologist to interpret them. The procedure is neither simple nor inexpensive and these are serious disadvantages. Patients who fail to appreciate the consequences of delay in the diagnosis of cancer may be reluctant to incur this expense.

Biopsy, being a surgical procedure, cannot be regarded as completely without danger to the patient. However, the direct operative risk is minimal. The surgical hazards are similar to those encountered in other operative procedures and include reactions to local or general anesthesia, infection at the operative site and hemorrhage. Hemorrhage is particularly apt to be a problem in fungating tumors of the cervix where the biopsy is taken from a mass of tissue too friable to retain hemostatic clamps or sutures. The same difficulty may be encountered on incising any soft or necrotic tumor mass which is too large for total excision. Biopsies on growths of this character are best done in a hospital, but biopsies of most superficial lesions can be done safely as a part of regular office practice.

A more serious criticism arises from the possibility that cutting into a malignant tumor may favor metastases, accelerate the local growth or disseminate tumor cells in the surgical incision. Hellwig[2] has reviewed the extensive experimental and clinical literature bearing on these problems and although he found no unanimous agreement it appears that the dangers are more imagined than real. Incision of a malignant neoplasm which is growing under tension may accelerate local growth but it is doubtful that clean incision with a sharp knife has any important effect in favoring metastases. In animal experiments massaging or squeezing neoplasms has resulted in early widespread metastases[3] while biopsy has had no significant effect.[4] It is quite probable that metastases in human cancer are much more likely to result from rough examination or vigorous palpation than from diagnostic incision. Reasonable precautions should be taken to prevent seeding of the surgical wound with cancer cells at the time of biopsy, but even if surgical technic is faulty in this respect, a pathologic diagnosis can be obtained within a few days and appropriate treatment instituted before tumor implants can make much progress. If surgical extirpation follows biopsy the entire biopsy wound should, of course, be excised with the surgical specimen.

Whenever a mass suspected of being malignant is accessible and of reasonable size it is preferable to remove the whole mass as a block to avoid the potential hazard of breaking into malignant tissue. This applies particularly to the removal of enlarged lymph nodes or nodules in the breast. In cases of suspected carcinoma of the skin excision of the entire lesion with adequate margins of normal tissue may accomplish both diagnosis and treatment in a single operation.

A proper use of pathologic diagnoses in the management of patients with cancer depends to a large extent on the clinician's familiarity with the pitfalls of the diagnostic method. A positive diagnosis of cancer made by a competent pathologist carries a very high degree of accuracy and is often the only means of establishing a diagnosis with any certainty. This is particularly true in early, curable lesions. A negative report on a biopsy is a very different matter. It by no means rules out the possibility that cancer is present but only indicates that no malignancy was found in the tissue submitted. Many cancers undergo extensive necrosis and frequently become secondarily infected. A small specimen taken from a tumor mass may actually consist only of necrotic debris or inflammatory granulation tissue. Sometimes a piece of normal tissue overlying a tumor nodule is submitted as a biopsy and the pathologic report is disappointing to the clinician. This accident is frequent in biopsies of lesions which undermine and elevate normal mucous surfaces as is often the case in carcinoma of the rectum or sigmoid. Here the obstructing lesion, as viewed through the proctoscope may be almost entirely covered by inflamed but non-malignant mucosa and a superficial biopsy will naturally be reported as showing no carcinoma. The accuracy of biopsy diagnosis depends as much on the care with which the tissue is selected and removed as it does on the skill of the pathologist.

Another point worth stressing is that pathologic diagnosis of tumors is not an exact science. It is similar in many respects to the process of clinical diagnosis and involves the weighing of evidence and the exercise of judgment. A pathologist serves as a medical *consultant* rather than a technician. Some surgeons still believe that they will get more reliable pathologic opinions, free of all prejudice, if they submit their tissue specimens to the pathologist without including any clinical information. The same men would feel hurt if they were called in consultation by an internist but were denied access to the clinical history or laboratory data. The purpose of supplying the pathologist with clinical data is solely to improve the accuracy of diagnosis. For example, the callus of a normally healing fracture can simulate very closely, under the microscope, an osteogenic sarcoma and a pathologist who is not on his guard can make a serious blunder. Metastatic carcinoma of the thyroid gland may reproduce almost perfectly the structure of normal thyroid but if the pathologist knows that the tissue came from a nodule in the skull he can make a useful diagnosis. A frequent problem in pathology is to detect metastatic carcinoma in lymph nodes. The tissue submitted is often scanty but the presence of epithelial cells in a lymph node is strong evidence in favor of carcinoma. Epithelial cells are not normally present in a lymph node but in a tonsil the structure of a lymph node is fairly well imitated and the surface and crypts normally carry a layer of epithelial cells. Without laboring the point it is easy to appreciate that although a clever pathologist can usually guess the source of a tissue and sometimes even the approximate age of the patient, such guesswork does little to improve the objectivity or accuracy of his diagnoses.

In this connection, I would like to make a point which is difficult to state fairly. Pathologic diagnosis is a valuable adjunct to clinical diagnosis but it is no substitute. If the pathologic report is obviously inconsistent with the clinical features of a disease it is a mistake to accept the pathologist's word as final. A second biopsy or a personal conference between the clinician and the pathologist will often clear up the difficulty. In the final analysis the clinician is still responsible for the diagnosis and care of his patient and the pathologic diagnosis should be treated only as an opinion, based on substantial evidence of a fairly trustworthy nature and not as final and irrevocable fact. It is quite obvious but somehow difficult to remember that the pathologist bases his diagnosis only on tissue removed from the patient's body while the clinician has to fear only the tissue remaining in the patient. A biopsy of the breast may be reported correctly as showing only intraductal hyperplasia but cancer may be present in another portion of the same breast.

Another common source of misunderstanding is the "equivocal" pathologic report. A surgeon may have gone to a great deal of trouble to obtain a biopsy and may have given his patient to understand that a definite diagnosis will result. His feelings are understandable when the report comes back, "Atypical epithelial hyperplasia, not diagnostic of malignancy." Such a report should be taken at its face value. It does not indicate that the pathologist is ignorant or uncooperative. It usually means that he is honest—he does not know—and is not afraid to say so. Conscientious pathologists realize the weight which is placed upon their reports and are conservative in making a serious diagnosis on insufficient evidence. I do not mean to suggest that pathologic reports should be ignored or discredited but only that they should be used intelligently, in conjunction with other evidence, in reaching a clinical diagnosis.

Turning to the more practical aspects of tumor diagnosis by biopsy a few points of technic are worth mentioning. Well preserved tissue is necessary for accurate interpretation and as soon as the specimen is removed from the patient it should be dropped into a jar of 10 per cent formalin or some other suitable fixing agent. Tissue which is partially dried, poorly fixed or decomposed is distorted and unsatisfactory for diagnosis. Similar distortion results from the removal of small biopsies with an electric cautery. The tissue margins are shriveled and burned beyond recognition. It is better to remove the tissue with a knife and then apply the cautery to the wound margins if necessary. When small skin lesions are excised for diagnosis it is often wise to remove the whole lesion. Most surgeons appreciate the importance of excising an adequate margin of normal skin about a lesion suspected of being malignant but they frequently fail to dissect deeply enough. The pathologist often discovers that cancer extends entirely through to the deep surface of a specimen and he must presume that some tumor cells have been left behind in the depths of the wound. In dissecting out enlarged lymph nodes for diagnosis the surgeon often encounters a group of nodes. Metastatic carcinoma may involve only one or two nodes in the group. The largest node is the one most likely to contain tumor and even though it may be more difficult to remove than a smaller node it is more likely to yield a reliable diagnosis. Inguinal lymph nodes are notoriously prone to show chronic inflammatory changes and fibrosis even in normal persons. In patients manifesting generalized lymphadenopathy the inguinal nodes are the least desirable for pathologic diagnosis.

A number of technics have been developed to simplify the process of obtaining tissue for pathologic study. Among these are the punch, aspiration or needle biopsy of such structures as the prostate and superficial lymph nodes and even of the lung, spleen, and liver. From the pathologist's point of view such biopsies may be adequate to justify an unequivocal diagnosis but often only a tiny shred of distorted tissue is available for examination and no positive report is warranted. Aspiration biopsies often give valuable information but are much less dependable than open biopsies.

Examination of peritoneal or pleural fluid and smears of bronchial or vaginal secretions may reveal malignant cells. Here the pathologist must rely on the characteristics of individual cells or small groups of cells. A diagnosis of cancer based on such evidence is less reliable than a tissue biopsy and failure to find malignant cells has little weight in ruling out the presence of cancer.

Frozen section diagnosis represents a great advance in the practical usefulness of biopsy. While the patient is on the operating table tissue can be removed and a section made, stained and examined within five to 10 minutes. The appropriate treatment can then be instituted without delay. In experienced hands frozen section diagnosis carries a high degree of accuracy but is still somewhat less dependable than diagnosis by routine methods. The slides are of relatively poor quality, the pathologist's decision is hurried and the surgeon must expect a higher proportion of equivocal reports.

Taking into account all the imperfections inherent in the pathologic diagnosis of cancer, biopsy still remains an indispensable and highly trustworthy method of determining the true nature of lesions suspected of being malignant. Pathologic examination of a representative piece of tissue will yield a definite diagnosis in the great majority of instances and there is no other method of distinguishing many benign lesions from early malignancy. Greater use of biopsies both in general practice and in the medical specialties will do much to decrease the death rate from cancer. No physician can escape the duty of prompt diagnosis of early cancer since here as in no other disorder "the more obvious the diagnosis the more hopeless the prognosis."

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DISCUSSION

Dr. Howard Mahorner (New Orleans): This paper is so important from the clinical standpoint that I can not resist the temptation to say something about how a surgeon feels toward a pathologic diagnosis, particularly one made on frozen sections. Many times the surgeon is put in the position of determining his action on the diagnosis of a biopsy. He must get back the diagnosis rapidly and must act on it rapidly. In medical centers, particularly, the most scientific method should be used to give that patient the best; and that means for lesions of the breast, lesions of the thyroid and lesions of the stomach, we know immediately whether there is a malignancy or benign lesion. Many times a small tumor of the breast is removed which is clinically benign and it turns out pathologically to be malignant. The only fair thing to do for the patient is immediate radical opertion. Under less modern conditions, that is, having to wait three or five days to have sections come through, the patient's chance for recurrence is increased. What I say may not enhance the pathologist's own opinion of his importance in the operating room. But I have the highest regard for the part the pathologist plays in determining what is done for that patient and I hope more and more pathologists will realize how much we rely on them, to tell us what to do for the patient. One of the most significant parts of this paper to me was that diagnosis of a biopsy is a consultation and not a laboratory procedure.

Dr. W. H. Harris (New Orleans): After a comprehensive and scholarly paper such as Dr. Dunlap has presented, there is little, if anything, for me to add.

Those of us who have occasion to receive biopsies mailed from remote areas are very often even more seriously confronted with what both Dr. Dunlay and Dr. Mahorner have indicated, namely the factor of a biopsy's primary purpose, which is consultation. We often receive minute specimens with no history and at times not even specified as to the area of removal. Not infrequently we are asked to wire a report. Under these circumstances where we can not readily communicate with the physician because of distances, such conditions are certainly not constructive, or to the best interest of the patient. A pathologist is pleased to do all within his capacity and especially happy to give encouraging information as rapidly as possible. It is naturally, however, exceedingly dangerous to make commitments where insufficient and essential data are not available. If, of course, the histologic picture of the specimen received is specifically and definitely malignant, one does not hesitate to give a telegraphic reply any more than for rush sections at the time of operations.

I am satisfied that with fixed tissues, frozen sections can be properly prepared and made for permanent record. There are, of course, various methods of staining frozen sections and it is quite possible, with a reasonable amount of care and a proper microtome to obtain frozen sections that will compare reasonably well with paraffin sections. It is needless to add that properly prepared sections embedded with paraffin or celloidin offer the best cytologic study and permit of special stains.

Some twenty-five years ago I had occasion to publish upon "The rush section as an aid to the surgeon." This occurred during the very active period of the former Presbyterian Hospital in New Orleans with a staff of such men as Drs. Parham, Denegre Martin, Jeff Miller, Clark and many other active men. Numerous rush sections were requested. In the preparation of rush sections a thin celloidin coating and rapid staining with the standard H & E method gives a very satisfactory slide for diagnoses.

I did not hear Dr. Dunlap allude to the subject of "potential malignancy." Such, of course, is a very loose term and often the surgeon asks "is it or is it not malignant?" However, if the pathologist considers that the histologic changes present a picture justifying the term potentially malignant, I feel that this forms a warning to the surgeon to observe carefully not only the reparative progress but the periodic postoperative progress.

I feel Dr. Dunlap's paper is a timely reminder of an important subject during this "high pressure" period wherin the expectancy from the pathologist remains great and the realization of coordination by clinician is apt to be overlooked. I have certainly enjoyed his excellent presentation.

Dr. G. H. Hauser (New Orleans): Just a few words to emphasize some of the important points which Dr. Dunlap has covered in his excellent presentation. In tumors that can readily be palpated, such as tumors of the breast, great care should be taken to avoid manipulation and undue pressure, which have been shown to be of far greater danger than section of a portion of the tumor for examination. It must also be remembered that a negative biopsy may be of little or no significance, especially in growths which are not readily accessible, such as tumors of the bronchi, larynx, or esophagus. Rush biopsies, in addition to their diagnotic value, can also greatly assist the surgeon in determining whether the edges of tissue about the growth are free of malignancy. This is of special value in malignancies involving the skin or superficial tissues, and also in benign growths which, if not entirely removed, may become malignant with recurrence of the growth.

Question—In smears, what do you consider characteristic of cancer, for example, the finding of epithelial cells?

Answer—That is a difficult question to answer. The recognition of malignancy is not a simple matter even in tissue sections and not something to be explained in a few words. Some one put it as being very much like recognizing one's grandmother—she would be hard to describe perfectly, but one can still recognize her.

In the diagnosis of smears we are up against the problem of recognizing cancer from the cytologic features of single cells. We must know the cells normally present in fluids from the different body cavities and note the presence of any abnormal cells. If an abnormal cell has a relatively large or irregular nucleus and scanty cytoplasm we are suspicious. If we are lucky enough to get a group of abnormal cells there is sometimes a mitotic figure or two in that group.

At best, I think the method of smear diagnosis of cancer is open to considerable error and should not form the only basis for a very radical surgical procedure unless it is confirmed by a second smear or biopsy and is in good keeping with the clinical features of the case.

CARCINOID OF THE APPENDIX WITH METASTASIS

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To retrace the historical background of carcinoids is not the purpose of this study, since Forbus stressed history in his excellent management of this subject in 1925. Rather will this report add to the ever increasing number of cases which suggest that carcinoids metastasize and are potentially malignant. Herein are presented three cases in which metastasis occurred.

To obtain a sizable number of cases on which to report, the records of Charity Hospital in New Orleans were surveyed for the years 1910-1945 inclusive. During that period there were filed 23 cases of carcinoids, all of which were of the appendix. The records at Mercy Hospital in New Orleans revealed six such cases between 1920 and 1945, also of the appendix.

In 1910 Huebschmann pointed out the similarity between the cells of carcinoids and those in the crypts of Lieberkuehn. His findings were further substantiated by Grosset and Masson, who brought out the fact that both of these groups of cells had a great affinity for silver compounds.

The belief that the cells discovered by Kultschitzky and named for him give rise to carcinoids is now indisputable. These cells are distributed throughout the intestinal tract: sparcely in the stomach and the colon; and in great numbers in the duodenum, jejunum, ileum, and appendix.

The origin of these argyrophilic cells has been a topic of disagreement among those who have shown interest in this subject. Raiford early postulated that they arose from ectoderm, and were to be found associated with nerves; further, that they had qualities common to other cells that arose from ectoderm. Danish's work on the chick embroyo showed that carcinoid cells arose from chromaffin tissue of the sympathetic gangelion, which in turn arose from ectoderm. They arranged themselves in rosettes similar to those of the neuroblastoma.

Masson supported the hypothesis that they arose from entoderm, since they were found in close association with the mucal glands. However, such an hypothesis is refuted by the fact that these cells do not appear in the mucosa until the third or fourth fetal month. Kull concluded that these cells arose from mesoderm and migrated to rest in the mucosa, which indeed sounds plausible.

INCIDENCE

These tumors occur more frequently in the appendix than in any other part of the intestinal tract. Porter and Whelan found that they occurred twice as frequently in the appendix, and that they were found in 0.2 per cent of the appendices they removed. Raiford stated that 85 per cent of carcinoids occurred in the ileocecal area. Jones believed they were found five times more frequently in the appendix as in the small intestine. Forbus stated that carcinoids were found to occur in 0.4 per cent of all appendices removed by operation. Raiford reported that 0.18 per cent of all tumors of the gastrointestinal tract were carcinoids. In a review by Simon of 139 cases, 77 per cent of the tumors occurred at the distal third, 14 per cent in the middle third, and 7 per cent at the base; 2 per cent involved the entire appendix. Of 40 cases reported by MacCarty and McGrath, 70 per cent occurred at the tip. During the periods surveyed in the records of the two hospitals mentioned above, I found no cases of carcinoids outside of appendix. Of the seven cases of carcinoids of the appendix in which the location was described, six arose at the tip and one at the base.

RACE, AGE AND SEX

Carcinoids of the appendix have been found most frequently in young individuals, averaging about 25 years of age, according to Raiford; and carcinoids of the other parts of the intestinal tract occur in individuals at about 55 years of age. This difference he explained by the fact that carcinoids of the appendix produced symptoms of obstruction sooner than the larger portions of the intestinal tract.

In the group of cases surveyed for this study 20 of the 29 cases occurred in persons between the ages 11 and 30. The oldest patient was 56 years old and the youngest was eight, the average age being 28 years, as compared with Raiford's average of 35 years for all carcinoids, and 25 years in his group of carcinoids of the appendix. In the local hospital survey the white patients accounted for 14 and the negroes for nine cases in the hospital where mixed admissions occurred; there were six cases in the hospital admitting only white patients. In the total group there were seven white males and thirteen white females; there were no negro males, but nine negro females. Thus, females accounted for 22 or 76 per cent of the entire group. In considering the various groups reported on, one might state that the females predominated over the males by a slight margin. However, that margin is diminished when one takes into account the greater prevalence of appendectomies among females due to pelvic surgery.

Although it is the opinion of Oberndorfer and Forbus that carcinoids are benign tumors, other very convincing evidence has been offered that they metastasize. Raiford reported that six cases of carcinoids found in the gastrointestinal tract outside the appendix were all malignant with evidence of metastasis. Cooke reviewed 104 cases of carcinoids of the small intestine; 48 proved to be malignant and showed metastasis. These findings were questioned by Raiford because the author did not state whether all the examined cells thought to be malignant had shown an affinity for the silver stain. Wyatt demonstrated that among six cases listed as carcinoids of the colon which he examined, three showed evidence of malignancy. In carcinoids of the appendix the path of metastasis is most frequently through the lymphatics to the regional nodes, as reported by Gaspor; and occasionally to the liver as reported by Gubitz. However, Marangas believes it is by infiltration of the blood vessels.

The cases studied in this survey brought to light the following facts. Diagnosis of these tumors is not made until the tumors have been inspected, often only by a pathologist. Among the cases where there was no metastasis only one surgeon made the diagnosis: in this case the tumor was large enough to palpate and had begun to involve the cecum. In only six of the cases diagnosed by a pathologist was the nodule identified beneath the submucosa and not apparently attached to the surrounding tissue. When they were sectioned, the cut surface presented a yellowish appearance. The size of the tumors varied from a few millimeters to centimeters. In some instances it is reported they produced an anular lesion simulating a carcinoma. Although these tumors sometimes occur in multiplicity there were no cases in the group under consideration where more than one tumor was identified. In the three cases reported as being malignant, metastsis occurred in the lymph nodes, in accordance with the findings of Gaspor and Raiford. A fibrous capsule surrounded the tumor, suggesting a host reaction.

The statements that carcinoids of the stomach are apt to become malignant more so than those of the appendix is erroneous. Those of the appendix become malignant and show their power of infiltrating the layers of the intestine, but before these tumors can cause further damage clinical manifestations of appendicitis arise, and they are extirpated. They are encountered in autopsy material of aged individuals with no more evidence of malignancy than those seen during routine appendectomies.

In ten of the cases herein reported the carcinoids had begun to infiltrate the tissue beyond it, and the serosa showed evidence of involvement. Carcinoids were associated with 14 cases of acute, or gangrenous appendicitis, 13 with obstructive appendicitis, causing recurrent pain. There were three cases in which there was a full-blown peritonitis resulting from ruptured appendix and in which a carcinoid played an important role.

The following three cases show definite evidence that the metastasizing lesion arose from a carcinoid of the appendix. They are taken from the hospital records already referred to:

CASE NO. 1

V. L., a negro female, 56 years of age, had been ill for one week prior to hospitalization, complaining of nausea, vomiting, distention, and fever. Examination revealed a tenderness and rigidity of the abdomen in the lower right quadrant. A diagnosis of appendicitis, with possible peritonitis, was made. Upon exploration a gangrenous, ruptured appendix was found with a large tumor involving the tip and fixed in the retroperitoneal tissue. The regional nodes were enlarged. The tumor, appendix, and nodes were removed. The pathologist returned a report stating that the rupture through the distal part of the appendix was due to a carcinoid tumor; the retroperitoneal tumor and node were malignantly related to the carcinoid and all the tissue examined took the silver stain.

CASE NO. 2

M. H., a negro female, 25 years of age, complained of pain in the lower right quadrant, with nausea and vomiting, which had been getting progressively worse for the preceding ten days. A diagnosis of acute appendicitis was made. Exploration revealed an acutely inflamed appendix with markedly enlarged mesenteric nodes. The appendix and the nodes were removed. The pathologist's report was: Carcinoid of the tip of the appendix with invasion of the neuroblastic cells into the submucosal and muscularis layers. The lymph nodes were infiltrated by cells that were morphologically related to carcinoids and which took the silver stain.

CASE NO. 3

C. B., a white female, 44 years old, entered the hospital complaining of pain in the lower right quadrant of the abdomen, particularly after eating. During the last few days she had become much worse. A diagnosis of subacute appendicitis was made. The appendix was acutely involved and was bound down by adhesions. Considerable difficulty was experienced upon delivering it. The pathologist's report was: Subacute appendicitis with a carcinoid obstructing the lumen at the junction of the distal and middle thirds. Four months later the patient was readmitted. She presented a mass in the epigastric area. Exploration revealed a great deal of fluid in the abdominal cavity. There was generalized seeding of the peritoneum and its contents. The pathologist's report was: Carcinoid involvement of the nodes.

SUMMARY

Twenty-nine cases of carcinoids were gathered in this survey, all of which were of the appendix. Three of these cases showed evidence of metastasis. There were ten cases that showed infiltration of the serosal layer.

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OTOLARYNGOLOGY IN GENERAL PRACTICE[*]

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We are living in a rapidly changing medical world. Progress in medicine has always been steady and certain but slow except on the occasion of the introduction of some valuable new diagnostic or therapeutic agent and then the changes become sudden and startling.

In otolaryngology a revolutionary change occurred in 1884 when the discovery of cocaine ushered in the specialty of rhinology by making possible painless intranasal procedures. Most of the early procedures have been abandoned or remodeled and many new ones have been introduced, but cocaine is still the number one drug on the rhinologist's table.

In modern times a similar abrupt revision of our therapeutic measures occurred with the introduction of the new antibiotic agents. The first major effect observed in clinical otolaryngology with chemotherapy was the elimination of most of the acute mastoid surgical procedures and since then miraculous cures have been reported in the more serious complications of all types of infections including cases of cavernous sinus thrombosis, a disease which formerly carried a 100 per cent mortality tag.

Along with the glowing reports of the achievement of the sulfonamides come the reminders that the drugs are toxic and the results of their use are often serious. More and more physicians are becoming aware of the dangers of indiscriminate and uncontrolled sulfonamide therapy. Davisohn,[1] pathologist at the Chicago Mount Sinai Hospital, recently sounded a note of warning against such practices and feels that even though certain compounds seem less toxic in some conditions they are all potentially toxic agents and may cause serious injury.

The toxic effects may be due to direct action of the drug on tissue or to hypersensitive reactions. They may be prompt, accelerated or delayed. The prompt or accelerated reactions occur mostly in patients who have received sulfonamides previously, the interval being days, months or even years.

Damage to the blood may affect one or more elements. Four conditions are observed. They are hemolytic anemia, leukopenia, thrombocytopenia and leukocytosis.

Hemolytic anemia may develop slowly, appearing late in the course of therapy or rapidly with a drop of red cells to the neighborhood of one million in 12 hours. In the severe acute type death is due not to the anemia proper but to renal failure. In these cases plasma is given to prevent shock and transfusions to restore blood values.

Sulfadiazine is less likely to cause hemolytic anemia than the other compounds but regardless of the drug used it should be discontinued if certain symptoms develop such as nausea and dizziness, pallor, fever, anemia, leukocytosis, increase in reticulocytes and renal disturbances.

Sulfathiazole is the least likely to cause leukopenia. This condition may occur with or without granulopenia. Agranulocytosis as a complication of the sulfonamides is less likely to prove fatal than the same condition caused by amidopyrine, the drug which caused so many deaths a few years ago. The white count should be checked in all cases and treatment stopped if it drops to 3500. The clinical picture shows a sudden turn for the worse with the appearance of ulcerative lesions in the oral cavity and on the mucous membranes and in extreme cases the prognosis is bad. Transfusions are given as are injections of pentnucleotide and liver extract with ample fluids.

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[*] Presented before the New Orleans Graduate Medical Assembly April 3, 1946.

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A number of cases of thrombocytopenia with purpura have been reported, the complication appearing early or late with a bad prognosis.

Leukocytosis with a count of 50,000 to 100,000 is occasionally seen and may appear concurrently with an acute hemolytic anemia.

Other conditions observed by the pathologists are periarteritis nodosa, all grades of hepatic damage, including cases of acute yellow atrophy with fatal outcome, and various types of kidney disorders.

Along with an understanding of the toxic reactions caused by the sulfonamides, the seriousness of their masking effect should not be overlooked. It should be known that even though mastoid surgery in acute cases has diminished, the serious complications of acute otitis have increased and these are observed at the autopsy table.[2] Symptoms which formerly guided the physician along the course of the disease are now clouded or masked entirely by intermittent doses of the sulfa drug. Now a child with a beginning ear infection receives sulfadiazine or one of the other compounds for a few days and the symptoms promptly disappear. The treatment is discontinued and the disease progresses. The drug is again prescribed, perhaps by a different physician. This routine keeps up while the infection travels quietly into the cranial cavity with a resultant meningitis or brain abscess. In the pre-sulpha days, drainage would have been established at the proper time in many of these cases through the ear drum or the mastoid cavity and the more serious complication would have been forestalled.

EARLY RECOGNITION OF CANCER

Early recognition of cancer is undoubtedly a manifestation of the highest type of diagnostic acumen and a physician with capabilities in this direction is one who is able to render a service of immeasurable value to his patients.

Unfortunately, most individuals are inclined to overlook or neglect the early symptoms of malignancy. They have a false impression that cancer is always associated with pain. This is, no doubt, due to their familiarity with suffering of those in the terminal stages. Because of this, they are apt to lose several valuable weeks or months while they ignore small superficial lumps, sores, enlarged glands or vague feelings of discomfort. Often the presenting symptom is an unnatural bleeding from one of the orifices of the body or a disabling functional disorder.

In the ear, nose and throat, early diagnosis of cancer is not difficult for one accustomed to examine these fields and who at the same time is cancer minded.

The oral cavity is readily accessable to direct inspection and palpation, the larynx indirectly with the aid of a mirror, the nasopharynx directly or with mirror, and in the nose, aside from anterior rhinoscopy, much information is gained by x-ray examination.

Early malignancy of the oral mucous membrane is evidenced by the appearance of a small, indurated painless plaque which infiltrates the membrane. Ulceration occurs early and may eventually spread to destroy a considerable portion of the lip, tongue or other structures. Metastasis is first, to the upper deep cervical and submaxillary lymph nodes. The enlargement of these nodes is often the first evidence of the disease, especially when the growth is located in the nasopharynx, pharynx or base of the tongue, areas not usually included in a casual examination.

Most cervical adenopathies in the adult are malignant and more than likely metastatic carcinoma from a primary lesion in the oral cavity or pharynx.

On occasions, infected tonsils have been suspected of being responsible for the glandular enlargement and their removal instituted forthwith. In some cases, according to Hayes Martin,[3] the tumor at the base of the tongue was so large that it was necessary for the surgeon to push aside the cancer in order to get at the tonsil.

The combined use of surgery, radium and x-rays is the treatment and the prognosis is not too discouraging, even in the advanced cases.

In the larynx, symptoms depend upon the location of the lesion. If on the true cords, hoarseness, of course, is the early and most important symptom; if in an adjacent but still intrinsic area such as the ventricles and below the false cords, hoarseness is a later development and if extrinsic, this symptom may be entirely absent or very late in developing.

The early symptoms of the intrinsic type are a feeling of discomfort, tickling sensation or a "lump in the throat" and later, pain on swallowing or talking. Some form of hoarseness is present but this may seem insignificant to the patient. It should not, however, be disregarded by the physician who should make sure that the larynx is examined with a mirror, even though the procedure is found to be difficult and require extensive local anesthesia.

It should be thoroughly understood that an early diagnosis is vitally important, for as is the case with most malignancies, treatment begun early is simpler and more effective. The management includes surgical removal or irradiation or a combination of the two.

There is a growing tendency on the part of laryngologists to rely more on surgery and less on irradiation in the treatment of laryngeal carcinomas. In certain single cord lesions, laryngofissure suffices; in others, total laryngectomy, and this is contraindicated only in those cases with inferior metastases or in those which have invaded the pharyngeal wall.[4]

Carcinomas of the nose may become quite large before causing marked discomfort and previous to erosion of the bony wall, few symptoms are present which should direct attention to this area.

Symptoms which appear early are pains along the distribution of the fifth nerve, noted especially at night, or on lying down. Patients often experience pains in the teeth or paresthesia with or without infraorbital pain for some time before seeking medical advice, and even then, before obtaining a proper diagnosis.

Much time may be consumed with removal or care of the teeth or with nasal and sinus treatments for a theoretical sinusitis. On occasions, growths are removed from the nose which appear to be polyps but which are, in reality, malignant.

A sound practice is to have all apparent polyps sectioned and studied. These growths are more than likely merely a manifestation of allergy or infection, yet the history of many cases of malignancy includes the removal of recurrent polyps.

A case observed by the writer in 1941 had had growths removed from his nose at various times during the preceding three years. Profuse bleeding always followed and it developed that the so-called polyps were in reality portions of carcinoma arising in the ethmoid. Complete removal followed by roentgen therapy and a radium pack, resulted in a cure which has lasted now over five years. In many instances, the malignancy is only suspected in the presence of external manifestations such as swelling over the cheek or displacement of the eye or when the growth has eroded through the nasoantral wall to become a bleeding mass in the nasal cavity.

An early diagnosis is often stumbled upon by routine examination of the sinuses. In certain films, suspicious areas require further investigation and the diagnosis may be reached by aspiration biopsy or direct biopsy through a Caldwell-Luc exposure.

The modern method of treatment is a combination of surgery and irradiation. The antrum is entered beneath the cheek and the walls of the cavity, including portions of the floor are removed, leaving a permanent opening through which radium may be placed subsequently. The operation is followed by protracted roentgen therapy and the prognosis in general is good.[5]

INTRANASAL MEDICATION

We began years ago in the gay nineties with oily sprays of various colors and flavors. These were worthless but fortunately, only slightly harmful. Then came the age of the nasal tampon, white or brown, depending upon your color preference.

The colloidal silver salts, neosilvol and argyrol are alkaline and irritating to nasal mucous membranes. They are harmful in the acute stages and of no value in the chronic. In subacute infections, their irritating action produces a flow of mucus which some think of value but the same could be produced by a few whiffs of snuff and much more could be gained in the treatment of these cases by using irrigation or the Proetz displacement method which draws out the accumulated secretions by suction.

We are now in the nose drop age. Nose drops came on with the introduction of ephedrine which temporarily shrinks down the nasal membranes. The more we use nose drops, the simpler we want them, the less we expect of them and the less we prescribe them. They afford relief for a few hours in cases of nasal blockage but have no curative value and their habitual use should be discouraged. They are also supposed to open up the drainage areas of sinuses but here again they are only a temporary expedient for home treatment until more effective therapy becomes available. If they must be prescribed, ephedrine 1 per cent in normal saline or some synthetic product such as neosynephrine serves the purpose.

The incorporation of sodium sulfathiazole in nose drops was unfortunate. Most of the preparations containing this drug are extremely irritating to the nasal mucosa; by and large they contribute very much to the promotion and continuation of nasal and sinus infections but at last happily are on the way out.

Solutions of tyrothricin and penicillin as nose drops are disappointing. Penicillin by inhalation is a new form of administration which is being widely used. The results so far in acute infections have failed to fulfill the expectations of its advocates.

MANAGEMENT OF CHRONIC SINUS DISEASE

At the outset I wish to state that sinus disease at the present time is not the hopeless condition it formerly was.

During the past 20 years considerable fine research and clear thinking have dispelled much of the confusion associated with the sinuses and thrown into the discard most of the antiquated multilating operations which did little more than disrupt nature's very fine mechanisms of defense.

We, at last, have come to realize the importance of the role played by the ciliated mucosa which lines the nasal and sinus cavities and the moving film of mucus covering it. We know that in the mucociliary mechanism we have a primary defense against infection, for pathogenic organisms picked up by the sticky mucus are destroyed. It is also known that in the event of a breakdown in this defense, an able secondary defense awaits the invaders in the subepithelial layers of the membranes.

Here through the capillary walls pour powerful inflammatory cells to attack the bacteria and carry them to the surface. In a sinus cavity, pus is thus formed and immediately waved toward the drainage outlet by the cilia.

In the early stages of sinusitis, the ostium is usually blocked by acute congestion and the pus remains in the cavity causing pain and toxic symptoms. As local immunity becomes established, the swelling subsides, the opening becomes patent and the exudate pours into the nose. In unhampered cases, the infection gradually diminishes and will heal spontaneously in a week or two, but often barriers are present which prevent free drainage and the infection lingers on. Should nothing be done to provide adequate drainage, a state of chronicity develops.

The proper treatment of a case of chronic sinusitis is naturally removal of the drainage defects which caused it. In many cases, simple infraction of the middle turbinate which has been blocking the opening, or the removal of polyps followed by a few irrigations will suffice. In others, more extensive procedures such as septal resection, control of an allergy or correction of other systemic disorders is required.

All patients require individual study and planning and sometimes a therapeutic regime covering a considerable length of time. And through it all, prerequisites of success are complete cooperation of the patient and an unwavering enthusiasm on the part of the physician. In the truly obstinate cases, extra drainage openings known as windows are required but rarely indeed, is the radical removal of sinus mucosa indicated and this should be instituted only with the understanding that the procedure offers no assurance of complete and permanent termination of the disease.

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THE MANAGEMENT OF COMMON EYE INJURIES IN GENERAL PRACTICE*

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This presentation will not be of significant interest to the eye specialists. It is elementary and planned primarily as an aid for the physician who is doing general work and is sometimes called upon to treat an injured eye.

Eye injuries are the most frequent eye conditions that the general practitioner is called upon to handle. Not only are injuries very frequently met with but they are the conditions that when handled correctly at the outset many complications may be avoided. What I hope to do is to give you a brief outline of some of the eye injuries met with and call your attention to a few points in their diagnosis and treatment.

Eye injuries might be classified broadly as extraocular (lids and walls of orbit) and ocular (eyeball itself); ocular foreign bodies may be considered separately as extraocular and intraocular; wounds, divided into penetrating and non-penetrating types.

Let's consider the lacerated eyelid. This is treated the same as any skin laceration except when the laceration is complete, involving conjunctival as well as skin surface. In these cases the skin and conjunctival layers are sutured separately, using fine atraumatic suture material, absorbable for conjunctiva and silk for skin. If the lid margin is carefully aligned and the above precautions are observed very little scarring will result and the development of entropion or ectropion is not likely.

Ecchymosis and emphysema frequently make their appearance in contusion injuries around the eye. They must be considered as symptoms of fracture of the wall of the orbit until proved otherwise. Many times fractures of the orbital wall are undiagnosed and result in noticeable deformities of face when the swelling subsides. Many are missed unless they are actually looked for but careful inspection and palpation of the rim of the orbit will usually reveal a fracture if there is enough deformity to warrant reduction. If there is any doubt x-rays almost always reveal the presence or absence of fracture. Frequently ecchymosis and especially emphysema appear following a blow to the eye and no other signs of fracture are present. This is due to a small fracture of the lamina papyracea of ethmoid and laceration of nasal mucosa. This requires only conservative treatment as the displacement is negligible.

Let us now begin the ocular injuries by considering chemical burns of conjunctiva which are commonly met with as a result of industrial and household accidents. When the conjunctiva is severely burned adhesions between the eyelids and eyeball (symblepharon) may occur. The immediate treatment is copious irrigation of the conjunctival sac with water. The eyelids must be completely everted and any foreign matter removed. Following this, thorough washing in cases of alkali burns with 5 per cent acetic acid, and in acid burns 3 per cent sodium bicarbonate is washed through the eye until all trace of the chemical is removed. Lime is best removed from the tissues with a 5 per cent neutral ammonium tartrate solution. If pain is severe it may be alleviated with ½ per cent pontocaine solution as drops into conjunctival sac. Cocaine should not be used as it is harmful to the epithelium. The adhesions previously mentioned form daily and have to be broken up daily until the surfaces heal. This is done by forcing the adhesion apart with a glass rod after having anesthetized the eye.

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Granuloma of the conjunctiva occurs most frequently when a foreign body has lodged beneath the conjunctiva, especially bits of wood or thorns. The granulomas manifest themselves as small pink masses polypoid in shape. If the granuloma and foreign body are removed the healing will be rapid and complete. I recall seeing a case recently of a trapper who said that several days previously he had stuck a piece of marsh grass in his eye and that he had gotten it out. He developed the granuloma several days afterward and this prompted him to see me. I removed the granuloma and just beneath the conjunctiva along the surface of the sclera a tip of grass was present. Healing was complete after its removal.

The cornea may be more or less involved in cases of conjunctival burns. In slight burns the cornea remains clear but stains with flourescein. Flourescein 2 per cent is a very valuable aid in detecting any break in continuity of corneal epithelium. One drop is instilled into the conjunctival sac, then after one minute it is washed out with normal saline and any erosion of the corneal epithelium will show up as a green stained area. Such burns usually heal within 24-36 hours and leave no scar. When the cornea is slightly opaque and stains deeply with flourescein some opacity is likely to result. In more severe cases the whole cornea may be destroyed. First aid treatment is the same as conjunctival burns but the deep corneal burns are likely to be followed by complications and will need treatment over a more or less extended period.

Ultraviolet burns of conjunctiva and cornea are seen most frequently in welders and are very painful with much burning, lacrimation and photophobia, the onset of which follows exposure to any ultraviolet light within four hours. The treatment consists of keeping the patient in a dark room with cold compresses over the eyes. The pain can be relieved with codeine and aspirin by mouth and instillation of drops of ½ per cent pontocaine or butyn solution into the conjunctival sac. Instill one drop of solution every three minutes until pain is relieved; this will have to be repeated when pain returns. Healing is usually complete within 24-48 hours and no permanent damage results.

Contusion injuries of the eye may range all the way from mere abrasions of the cornea to rupture of the eyeball itself.

Corneal abrasions are commonly seen following a glancing blow on the cornea. A frequent example is the case where the child hits the mother's eye with its fingers; another is the abrasions of cornea of infants when they roll over in the crib and an edge of the starched sheet scratches the cornea. Abrasions are a source of great concern as they are very painful and accompanied by much photophobia and lacrimation. When flourescein is instilled into the eye the lesion is readily seen as a green stained spot; even without aid of flourescein the depolished area of cornea can nearly always be seen. Healing is usually complete in 24-48 hours; but occasionally the newly formed epithelium will become eroded and the eye will again become painful until it heals over again. The treatment is directed toward relief of pain until healing takes place. The eye should be kept bandaged and pain relieved by instillation of pontocaine or butyn ½ per cent. Anesthetic drugs, especially cocaine, delay the healing of corneal epithelium and should be used as sparingly as possible.

Hemorrhage into the anterior chamber (hyphemia) follows more severe contusions. The blood can be seen settling into the lower portion of the anterior chamber. If the hemorrhage is small in amount it will usually be absorbed quite rapidly but if it fills the entire anterior chamber glaucoma and other complications may set in. In the more mild case the patient needs only to be kept at bed rest and the eye bandaged until absorption is complete. Rest in bed cannot be over-emphasized in these cases as severe hemorrhages can take place 48-72 hours after a contusion injury which originally produced only a mild amount of hyphemia. The patient must rest until absorption is complete as physical exertion may cause more bleeding to take place. The coagulant drugs such as koogamin and synkamin may be used as an added safeguard against extra hemorrhage. A 1 per cent solution of pilocarpine may be used as drops (one drop three times daily) to guard against a rise in intraocular tension. Atropine should not be used.

Iridodialysis and traumatic mydriasis are conditions which occasionally follow rather severe blows to the eye. They are usually permanent and no treatment is indicated unless some other existing condition demands treatment. Iridodialysis is a rupture of the root of the iris resulting in a peripheral defect through which the red fundus reflex can be seen. Traumatic mydriasis is a permanently dilated pupil due to rupture of the sphincter muscles of the iris; sometimes the pupil is dilated due to paralysis of the nerves to iris sphincter and this type may recover to a certain extent.

Traumatic cataract, hemorrhage into vitreous, rupture of choroid, edema of retina and rupture of eyeball occur in severe contusion injuries. These usually affect vision to a more or less great extent and usually require protracted periods of treatment.

FOREIGN BODIES

Foreign bodies of the eye are very common and may be classified as extraocular and intraocular. Very frequently a small particle of foreign matter such as cinder will blow into the eye and this may become adherent to the conjunctiva (quite frequently under the upper lid) or cornea and may be removed with a cotton tipped applicator. A more difficult type to handle is the small steel or rust particles frequently found in welders and mechanics. These usually are buried rather deeply into the surface of the cornea. They are removed with a corneal spud after use of local anesthetic in form of butyn or pontocaine drops. A very important thing to keep in mind here is the so-called "rust-ring." This is the brownish deposit left on the cornea after the foreign body has been removed. This must be removed or the patient will continue to have trouble with the same symptoms as before removal of the foreign body.

Intraocular foreign bodies and perforating wounds of the eyeball may be considered together. As in all perforating wounds the possibility of an intraocular foreign body must be kept in mind even though it can frequently be ruled out on history alone. X-ray examination of the orbit should be done where foreign body is suspected. Since all perforating wounds require hospitalization and frequently surgery the first aid or immediate treatment consists of recognition of the ocular perforation, instilling atropine and protecting the eye with a bandage.

Most important is recognizing the presence of a perforation of the eyeball or the presence of an intraocular foreign body, which is not always as easy as one might think. For emphasis of this point I will cite a case which I recently observed. This patient was a mechanic who was working on a tractor and had his head under the hood while the motor was running. He felt something hit his eye but after a few minutes it no longer bothered him. The next afternoon he reported to the company doctor because his eye had begun to pain him and was slightly red. Upon examination the doctor found no foreign body and no perforation of the eyeball. Nothing significant was seen other than a slight injection around the limbus. The patient was dismissed with instructions to return the next day if the eyeball was still red; it was and the patient was sent in to me. Upon gross inspection of the eye the only significant findings were the injection around the limbus and a slight irregularity of pupil. There was no visible perforation, the anterior chamber was full and there was no decrease in intraocular tension. The fundus was normal and there were no vitreous or lenticular opacities. Upon slit lamp examination, however, some cells were seen in the aqueous and a slight amount of exudate was seen on the root of iris at 6 o'clock. On the strength of these findings an x-ray examination of the orbit was made; the plate plainly revealed a small foreign body in the eyeball. I mentioned this case to show how few findings can be present in cases of perforations and intraocular foreign bodies.

Usually, however, a wound in the cornea or sclera can be seen. Other signs of penetrating wounds are: marked reduction in intraocular tension, prolapsed uveal tissue or vitreous and a shallow anterior chamber if the wound is through the cornea. The prognosis of all perforating ocular wounds must be guarded because even when best results are secured there is generally a considerable loss of vision. Of course, if infection should develop, even the very smallest wound may lead to loss of the eye. These cases are therefore definite surgical emergencies and the sooner the proper therapy is instituted the better the prognosis.

In general the treatment of perforating ocular injuries is: removal of foreign body if present, excise prolapsed tissue then suture the wound or cover the defect with a conjunctival flap, to prevent and treat infection.

I hope this outline will enable you to differentiate the serious from the less serious eye injuries and help you to handle with more confidence eye injuries in general.

DISCUSSION

Question—How did you handle the motor mechanic in whom you found the foreign body in the globe?

Answer—We of course hospitalized him and put him on penicillin; put atropine in the eye to keep the pupil dilated and reduce iritis. We also localized the foreign body which was found to be in the root of the iris. It did not puncture the lens and no cataract developed. We are checking from time to time to see if there is spread of iron granules throughout the eye. The man still has the foreign body. We first combated infection with penicillin and later on sulfadiazine and fever therapy. When the inflammatory process cleared we found no bad result from the foreign body and left it in. We are observing occasionally for complications which might warrant removal of the foreign body.

Question—Do repeated welder's arc burns ever cause any permanent damage such as inflammation of inside of eye or cataract?

Answer—Welder's burns or arc burns, ultraviolet light burns etc., when repeated quite often do not cause any permanent damage to the eye. Ultra-violet rays are absorbed by the cornea, iris and conjunctiva and do not reach lens or the vitreous or the retina.

Question—Discuss the use of yellow oxide of mercury merthiolate ointment, and penicillin ointment.

Answer—Yellow oxide of mercury ointment is used quite frequently in conjunctivitis, and in styes and you seem to get pretty good results in styes along with hot compresses; hot compresses do more good then yellow oxide of mercury.

Penicillin ointment works very well in conjunctivitis cases especially gonococcal and pneumococcal and also in infection. I had one bad experience with penicillin ointment and I believe this was in the first case I ever used it on. The man came in the office with an early sty on his eye and was leaving town that day. I thought I would be smart and used penicillin ointment. He got the ointment and went to Kansas City and when he got there he really had an eye. He went to a doctor up there and he said "what have you been using? He said penicillin ointment and the doctor said get off of that and you will be all right. The point to bring out is it seems there have been quite a few patients who used penicillin ointment and got an allergic reaction; it is still good though in eye infections.

As far as merthiolate is concerned, I have not used much of that myself except in several cases where there have been chemical burns of the conjunctiva. I use it in the eye every three hours and in cases in which I have used it it worked very well.

Question—How is the rust ring removed?

Answer—In most cases where you have a rust ring remaining after a foreign body has been in the cornea you can remove with eye spud. Other men use a small dental bur and you can fit the dental bur into the ring and bore or turn it a little and that will also remove the rust ring.

Question—How would you treat an eye that was infected by an intraocular foreign body that you had previously removed?

Answer—At the first sign of infection in the eyeball you want to pour on everything you can because infection in that eyeball is really dangerous as far as sight is concerned and of course if not already in hospital you will want to get the patient in quickly and dilate the pupil with atropine. Start on penicillin 20 to 40 units every three hours; also give this penicillin with sulfadiazine — adequate sulfadiazine; 15 grains every three or four hours. It is also a good idea to give the patient some fever therapy in order to increase body resistance in general. A severe infection in the eyeball won't give you any fever at all and of course producing fever is a good way to combat infection in the eye. That is the way the body defends itself from infection; induce fever on that patient. A good way to do this is to give some typhoid vaccine, intravenously, about 25 million killed bacteria. Kick fever up every two or three days and also you can put hot local applications to the eye to increase local resistance. I think those are the high points in treating intraocular infection.

RH FACTOR AND ERYTHROBLASTOSIS FETALIS

A CASE REPORT

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In recent years there has rightfully been an increasing interest shown in the Rh factor in obstetrics and its related pediatric problem, erythroblastosis fetalis. Some of this interest has been abated, however, by the multiplicity of terms used in discussing the problem, and also by the fact that few laboratories were able to determine Rh factor accurately. There has also been considerable confusion among both general practitioners and specialists as to just what the Rh factor is, and in which individuals it was apt to cause complicacations.

The Rh factor is a newly discoverd factor of human blood first demonstrated by Landsteiner and Weiner[1] in 1940. It has been shown that this factor is found in 85 to 87 per cent white humans, 93 to 95 per cent negroes and in over 99 per cent Chinese and Japanese[2]. Those who have this factor are said to be Rh positive. Those who do not have this factor in their blood are Rh negative. It is only the combination of Rh positive father and Rh negative mother that is of clinical significance at present. It is only this combination that produces erythroblastosis fetalis in the offspring, but only about 5 per cent of these combinations result in sensitization reactions producing erythroblastosis. This sensitization by the Rh factor follows the familiar pattern of any bacterial immunization or sensitization. Those mothers who show an absence of Rh may be sensitized by pregnancy or by transfusion with Rh positive blood.

The presence of the Rh positive baby in utero causes the formation of anti-Rh antibodies in the blood of the Rh negative mother, or the transfusion of Rh positive blood into an Rh negative woman will cause the same result. The initial pregnancy or transfusion only serves to sensitize the mother and usually does not give any unusual results. It is the subsequent transfusion or pregnancy that gives us trouble. The end result of the sensitization is hemolysis, but in testing it is observed as an agglutination. Levine[2] has recently shown that some Rh negative mothers are incapable of producing complete antibodies that can be shown to agglutinate Rh positive blood, but do develop an incomplete antibody which specifically unites with and coats the surface of the Rh positive blood cells. This specifically coated Rh positive blood is incapable of reacting with potent anti-Rh agglutinogens. These are the so-called blocking antibodies of Weiner. The end result is the same, however, and erythroblastic babies may come from these mothers too.

Only in recent months has a testing serum of sufficient potency been generally available. Until this more potent serum was developed by Hill, et al.[4] there had been considerable discrepancy in the results obtained from checking suspected Rh factor sensitizations. In our laboratory using one type of testing serum we might find a patient to be Rh negative and demonstrate antibodies in one titer and sending the patient's blood to other serologists for checking, one might confirm our results and the other might get opposite results. Since using the new serum, our results have been uniform. However, to add to the confusion Kennedy[5] has shown that all cases of erythroblastosis are not found in families with the classical combination of Rh positive father and Rh negative mother. Some of these are probably due to sensitization by sub groups of Rh or M or N factors.

I feel that every pregnant mother should be checked for Rh factor early in pregnancy. If she is found to be Rh negative the father and any children should be checked. If the father is found to be Rh positive I check the mother at regular intervals for antibodies. If, after the baby is viable, an increasing titer of antibodies is found I feel the baby should be delivered by section. Also, all females should be checked before transfusing and if Rh negative, should only be given Rh negative blood. This would avoid many embarrassing reactions from transfusions and might save the woman trouble with future pregnancies.

The following case is reported because of the unusual factors found and also in the hope that others may profit by the mistakes we made, even though we were successful in obtaining a normal baby and healthy mother.

CASE REPORT

Mrs. S. B., a white female, aged 23, was first seen on March 14, 1944, para 1, gravida 2. She gave a history of her first baby being stillborn on August 29, 1943 which was delivered in another hospital and was told the cause of stillbirth was faulty attachment of cord and that the cord was very short. Present pregnancy showed L.M.P. October 20, 1943 with E.D.C. July 27, 1944. She had an uneventful prenatal course until June 23, 1944 which was her last visit to the office. At this visit the blood pressure was 110/80, F.H.T. 160, baby L.O.A., urine negative. On July 5, 1944 she noted absence of fetal movements and within a few hours went into labor. On admission to hospital no fetal heart tones were heard on repeated examinations. On July 7, 1944 she was delivered of stillborn female infant under continuous caudal analgesia. This fetus showed definite evidence of erythroblastosis. No Rh studies were done prior to delivery as we were not running them in our laboratory at that time. In October 1944 examination revealed the mother to be Rh negative and father Rh positive, but no antibodies were demonstrated in mother's blood. Contrary to our advice she became pregnant again and came to the office on September 14, 1945 with history of L.M.P. May 18, 1945 and E.D.C. February 25, 1946. At this time one serologist was unable to demonstrate anti Rh agglutinin in her blood, but two others were able to; one in titer of 1:20 and the other in titer of 1:4. She again had an uneventful prenatal course and repeated checks failed to show any increase in the titer. It was decided to deliver her by section and this was done by Dr. Marvin T. Green on January 29, 1946 under continuous caudal analgesia. The baby was a girl with birth weight five pounds, two ounces. Color was good, no evidence of edema or cyanosis and cried lustily on delivery. She was given 15 c.c. of Rh negative O blood in cord vein immediately, placed in incubator and given continuous oxygen. Blood count was red blood cells 3,925,000, hemoglobin 65 per cent. On January 30, she was given another 15 c.c. Rh negative O blood I.V. On second day the baby developed rather severe jaundice which increased in severity until given another 12 c.c. transfusion of Rh negative O blood, following which the uterus completely cleared. Daily blood examinations showed a progressive anemia starting on the third day and lasting until the nineteenth day, when it began to climb. This anemia was in spite of giving the repeated transfusions. On the sixteenth day we started giving the baby blood intraperitoneally, giving 30 c.c. daily for four days. This was done because it became practically impossible to enter the baby's veins. Only after giving blood in this manner did the blood picture improve. The birth weight dropped to a low of four pounds, twelve ounces before starting to gain. The baby was placed on powdered milk formula supplemented with vitamin C, iron, and cod liver oil. It was also given vitamin K for the first week. The baby was discharged from the hospital February 20, 1946 with weight, six pounds, two and a half ounces, red blood cells 2,700,000 and hemoglobin, 50 per cent. It has continued to do well and when last seen on May 6, red blood cells, 4,100,000, hemoglobin, 65 per cent, weight 12 pounds, four ounces. The mother had an uneventful convalescense. Other than the jaundice and anemia the baby showed no other signs of erythroblastosis. The baby's blood is Rh positive A.

DISCUSSION

With knowledge gained from this case and recent literature[6],[7] we feel that our biggest mistake was in not giving larger initial transfusions. If the baby had been given 60 to 75 or 100 c.c. of blood daily for the first three days it probably would not have had such a marked anemia. Of course it is questionable whether this larger volume would have embarrassed such a premature baby's vascular system. As was done in this case, we feel that the baby should not be allowed to nurse the breast as the antibodies are carried in mother's milk. Also, the short time interval between the pregnancies increased the chances of having trouble. As Davenport[8] has stated, at least two years should elaspe between pregnancies in sensitized women.

Another point of interest is that apparently this baby's hemopoietic system was stimulated as much by the intraperitoneal administration of blood as by the intravenous method. In a premature baby this is of value due to the difficulty in entering the very small veins of such a baby. In this baby the veins seemed much thinner than those of the usual baby. Whether or not this is a characteristic of erythroblastotic babies I am unable to say. The question also arises of how long to administer Rh negative blood to an Rh positive baby. I feel that Rh negative blood should be given as long as the baby requires blood. Apparently the antibodies are still present and would only serve to hemolyze Rh positive blood if such were given.

I want to express my appreciation to Dr. J. W. Davenport, Jr., of New Orleans, La. and Dr. W. R. Mathews of Shreveport, La. for their help in checking the blood of this patient for us and for their counsel in the handling of the case.

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PENICILLIN IN ACUTE BACTERIAL ENDOCARDITIS

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As a result of the cure of a patient with acute bacterial endocarditis treated with massive doses of penicillin, our interest was stimulated in the value of penicillin therapy for this disease.

In reviewing the literature it was found that forty-one patients[1-14] with acute bacterial endocarditis have been reported as treated with penicillin. Sixteen of these were described as cured. According to the etiologic organism, the cured patients may be listed as follows: eight with staphylococcic endocardititis, [1-4] four with pneumococcic endocarditis, [5-7] and four with streptococcic (Hemolytic type) endocarditis, [1, 6, 8]. In those in which the dosage of penicillin was stated it ranged between 960.000 and 58,640,000 units. To evaluate the role of penicillin in the treatment of a hitherto uniformly fatal disease, one must know as much as possible about the clinical status of the patient and must have an equally detailed account of the management. With some exceptions this was not given in the available reports. It is hoped that more details will be presented in the future.

The overall mortality was 39 per cent. This is high, and without critical examination of these reports gives a false impression as to the value of penicillin in the therapy of this disease.

Keefer[13] reported nine patients with endocarditis due to *Staphylococcus aureus* treated with penicillin with a 100 per cent mortality. The dosage of penicillin varied between 270,000 and 2,340,000 units administered over periods of time varying from four to 52 days. He summarized the results by stating that many were treated late in the course of the disease and the dosage was inadequate. Glaser, Smith et al[4] report six patients with *Staphylococcus aureus* endocarditis, one of whom died with rupture of a brain abscess after five days' therapy. Dawson and Hobby, [10] in discussing penicillin therapy, reported three patients with pneumococcic endocarditis in whom penicillin therapy was begun with great improvement resulting initially. In two of these patients the blood stream was temporarily sterilized but because of sub-

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sequent unavailability of the drug, penicillin therapy had to be abandoned. All three succumbed. One of Herrell's patients[9] with *Staphylococcus aureus* endocarditis died on the first day of penicillin therapy. The causes of death in Dolphin and Cruickshanks' study[1] were as follows: one patient with *Hemolytic streptococcus* endocarditis died six months later of heart failure; another died 46 days after penicillin therapy of bronchopneumonia following a mid-thigh amputation, and a third with *Staphylococcus aureus* endocarditis of uremia on the twenty-first day of the disease although the blood culture had become sterile. One of Meads' patients[6] with *Hemolytic streptococcus* endocarditis died after two intramuscular injections of penicillin.

This analysis shows that other factors accounting for death were present in many of the patients which in no way could be attributed to a failure of the drug, and although the mortality does seem high on casual inspection, it is not a true picture of the value of penicillin in the treatment of acute bacterial endocarditis.

REPORT OF A CASE

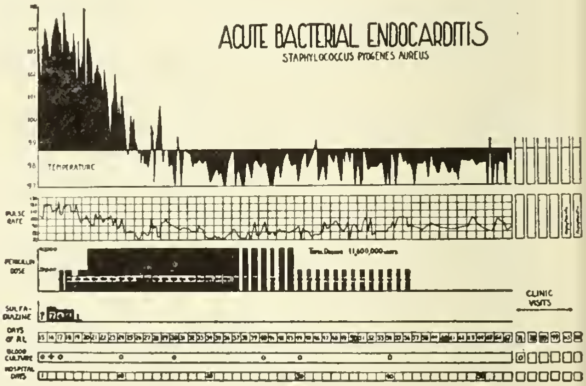
Present Illness: The patient, a 25 year old colored female, was admitted to the Charity Hospital at New Orleans the morning of September 2, 1945 with the chief complaint of severe pain in her neck. This pain started suddenly the night of August 31, 1945, approximately 36 hours before admission. At the time of admission she was almost moribund but was able to answer some questions. The pain radiated from the neck to the top of her head posteriorly.

The present illness dated back two weeks, during which time she had been treated by her local physician for chills and fever with accompanying left thoracic pain, and daily epistaxis. A course of sulfonamides was administered during the week previous to admission. Nausea and vomiting were associated with the onset of the neck pain. The left middle finger was swollen and painful. For three weeks preceding the acute episode, she had a furuncle on the left elbow.

Past History: Since early childhood there had been frequent episodes of tonsillitis. Tonsillectomy had been advised on several occasions but refused. In 1940 her first child was delivered by cesarean section. In 1941 she had a normal delivery. During the summer of 1944 she experienced severe joint pains and was admitted to Charity Hospital and a diagnosis of rheumatic fever with mitral stenosis was made. This attack was characterized by a painful, migrating arthritis accompanied by subcutaneous nodules over the involved points and distal portions of the limbs. There was cardiac enlargement and apical systolic and diastolic murmurs. After salicylate therapy and strict bed rest for four weeks she was discharged as much improved, but advised to remain in bed.

Physical Examination: At the time of the present admission the patient was found to have an enlarged, heaving, rapidly beating heart with a loud apical systolic murmur. A small hemorrhagic area was found on the left sclera and characteristic petechiae on the tips of several fingers and the soles of the feet. Her neck was moderately stiff. There were no other abnormal findings.

Laboratory Examinations: The hemogram showed: white blood cells 10,200 (87 per cent polymorphonuclear leukocytes), red blood cells 2,800,000, hemoglobin 70 per cent, sedimentation rate (uncorrected) 41 mm. Spinal fluid examination, September 2, 1945, revealed a pressure of 194 mm. of water, many crenated red blood cells, 672 mgm/100 c. c. of chlorides, 54 mgm/100 c. c. of glucose and 290 mgm/100 c. c. of protein. The supernatant fluid was xanthochromic. The spinal fluid culture was negative. On September 3, 1945 the spinal fluid revealed a pressure of 230 mm. of water and numerous red blood cells and on September 9, 1945 it was clear with a pressure of 366 mm. of water. Blood culture made the day after admission showed numerous colonies of *Staphylococcus pyogenes aureus.*



The hospital course is illustrated by the chart. It can be seen that a total of 11,300,000 units of penicillin were administered intramuscularly. There was little or no clinical response until the penicillin was increased to 480,000 units per day (40,000 units intramuscularly every two hours, day and night). The blood culture became negative after 120,000 units. She remained under observation in the hospital for 53 days and six months in the out-patient-clinic. Other than for the physical findings of mitral stenosis, which she had before her present illness, she continues to be well to date.

COMMENT

This case fulfills the clinical criteria for a diagnosis of acute bacterial endocarditis with cure following the administration of penicillin. From this experience and that of others, it appears that when penicillin is administered early and adequately the mortality of this disease can be materially reduced. The organism should be isolated, its sensitivity to penicillin determined *in vitro* and adequate dosage administered early. It is our opinion that adequate dosage is at least 0.5 million units per day. There have been only four patients reported in the literature who have received penicillin dosage comparable to this amount[3, 4]. These patients and the one now being reported had uneventful recoveries.

Early and large dosages of the drug is in line with the present day concept of penicillin therapy in subacute bacterial endocarditis. Although there have been cures in acute bacterial endocarditis using smaller doses than 0.5 million units daily, the presence of highly sensitive strains of organisms may have accounted for the good results. In general, small inadequate doses are to be condemned as they may result in penicillin-fast strains. It is quite likely that even larger dosages will prove to be better. The use of measures to increase and maintain high penicillin levels in the blood may prove to be of further value.

SUMMARY

A patient with acute bacterial (staphylococcic) endocarditis is described in whom fairly large doses of penicillin effected a dramatic clinical cure. The value of penicillin therapy and dosage are also discussed.

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PAROXYSMAL AURICULAR FIBRILLATION

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Formerly spoken of a "perpetual arrhythmia", auricular fibrillation is now known to occur in transient form as paroxysms of irregular tachycardia. The paroxysmal form of the disease is also more common than once was thought, as its appearance in a multitude of systemic and cardiac diseases as well as in normal individuals has become recognized with increasing frequency. Taxonomologically, the disorder may be placed with those cardiac conditions characterized by a disturbance in the cardiac mechanism, but differs from all of them in that, in itself, is is not evidence of cardiac disease.

In order fully to understand the findings, clinical and electrocardiographic, to realize the various roles which the etiologic conditions play in its occurrence and, above all, rationally to treat it, the underlying cardiac mechanism and the physio-pathology by which paroxysmal auricular fibrillation is induced must be understood. Like auricular flutter, auricular fibrillation, both permanent and paroxysmal, is produced by a disordered initiation and transmission of the impulse which results in exceptionally bizarre and rapid auricular action. The belief held by most cardiologists at present is that it can be explained most readily by the concept of circus movement, first demonstrated by Mayer and 1908, rather than on the basis of ectopic foci of impulse initiation, the hypothesis long held in Europe.

The experiment first performed by Mayer to demonstrate the existence of circus movement was to show that an electrical impulse applied to one point on the periphery of the umbrella of a jelly-fish (Medusa) would cause a depolarization wave to pass over the umbrella which would contract in its wake, then lie quiescent as the tissue underwent repolarization. If, however, he produced a local block in one small area of the periphery and applied the stimulus again, the contraction wave failed to pass the blocked area (which was then refractory) but would continue about the periphery of the umbrella in the direction opposite from the block, and, finding excitable muscle continuously in its path as it made the circuit of the umbrella edge, would proceed until it reached the previously blocked area. If this block had now become once more excitable, the depolarization wave, upon reaching it, would stimulate it to contract and pass on beyond it into the area from which it started. The refractory phase of the muscle was, however, such that during the circuit of the umbrella, by the depolarization wave the point of stimulation had once more become excitable, as had the blocked area, with the result that the wave, continuing to find excitable muscle in its path once more started the circuit without further stimulation being applied and was able to repeat its path indefinitely. Garrey[4] and Mines[1] linked up Mayer's observations upon the jelly-fish with the fibrillation of cardiac muscle. Mines showed that similar circular motion of the contraction wave could be induced in the cold-blooded heart, and Garrey that these waves could be produced in auricular muscle long as there was sufficient amount of tissue present to allow the refractory period to be overcome before the head of the circulation wave once more reached its starting point.

Lewis[7] studied this movement in mammaliam muscle tissue and concluded that, as in Mayer's Medusa, local blocks also existed during the condition in heart tissue. These blocks were described as being produced by the partial refractory state of isolated muscle islands.

From these investigations and others, it was concluded that four conditions were involved in the establishment of the circus movement: (1) local blocks in the auricular muscle; (2) a shortened refractory period; (3) a slow rate of conduction of the wave and (4) an annular course of sufficiently large diameter to permit a complete circuit to become propagated.

That clinical flutter and fibrillation are brought about by the induction of a circus movement within the auricle is subject to little doubt. The impulse is believed to travel in the natural rings of muscle encircling the openings of the great veins; this has been demonstrated electrocardiographically by Lewis[7]. This circular wave is the governing factor in flutter rhythm, from it centrifugal waves are thrown off into the tissue adjacent to the circular pathway from which they proceed through the auricular musculature and die away in the outlying regions: the right and left appendiges and the sleeves of the great veins.

Flutter and fibrillation are now considered to be of essentially the same nature and impure flutter the transition between pure flutter and fibrillation. In pure flutter the circular wave is repeated over and over again with precise accuracy, and the centrifugal waves pursue their prescribed courses uniformly from cycle to cycle, setting up rhythmic contractions of the auricles at a rate between 200 and 350 per second. In impure flutter the irregularity is accounted for by the greatest degree of deflection of the centrifugal waves brought about supposedly through an increase in the partial refractory state, that is, more unexcitable fibers in the path of the waves in their passage through the eccentric regions of the musculature with change in their pathways from cycle to cycle. In fibrillation, though the general revolving movement of the central wave is still maintained, it does not follow the same course from cycle to cycle and the centrifugal waves are also deflected from one path to another, both for the same reason: the presence of more islands of refractory muscle in the pathways of these impulses. Aside from this deviation in the pathway of the main waves by these islands, they also disturb the regularity of contraction in another manner. These islands of muscle tissue though refractory when the impulse first strikes them may become excitable while the impulse is passing around them and thus become excited in a retrograde manner, and, possibly, setting up other circus movements of a shorter duration and more temporary pathway. The net result is that there is at all times within the auricle depolarized, repolarizing and excitable muscle so that the organ is never still but is with some few fibers in a contracted state at various places throughout at a given instant. This produces an organ whose walls are quivering at a rate of from 400 to 550 contractions per minute and are distended by the contained blood which the asychronous and multitudinous contractions are unable to expel.

This mechanism prevails in both permanent and paroxysmal auricular fibrillation. Though permanent auricular fibrillation is usually associated with organic disease of the heart, paroxysmal fibrillation is not, surprisingly, necessarily so. In a series of 200 cases of paroxysmal auricular fibrillation, Parkinson and Campbell[10] found the occurrence of the disorder to occur in patients with detectable organic heart disease in 85 per cent, while in 15 per cent no evidence of heart disorder other than the paroxysms could be detected clinically or electrically, nor was there any systemic disease which might affect the heart nor any history of an etiologic factor known to produce heart disease.

It is evident that auricular fibrillation, permanent and paroxysmal, with heart disease or without it, can be produced by any condition which is capable of causing local blocks to appear in the auricular muscle and depressing the conduction time of its fibers. As a general rule, only conditions which actually produce a permanently diseased state of myocardial muscle will cause the condition to become permanent. On the other hand, however, almost any condition which is capable of producing temporary changes in the physiologic state of auricular muscle, whether these changes result in permanent damage or not, is capable of producing paroxysms of auricular fibrillation. Thus the disorder may be encountered in acute rheumatic fever, in thyrotoxicosis, in mitral stenosis before it takes on its permanent form, in hypertensive heart disease, in pneumonia, "chronic myocarditis," in coronary occlusion and angina pectoris, as well as in a host of apparently unrelated disorders such as chronic gallbladder disease, angioneurotic edema, after or during general anesthesia[8] and in persons with no heart disease whatsoever.

The mechanism by which the necessary changes appear in the auricular muscle in people with organic heart disease or in those diseases which are capable of producing slight physiologic aberration through toxic effects (as in pneumonia and any severe infection) or by virtue of increasing the strain on the heart (fevers of almost any sort, for example) is at once obvious, that by which the changes occur in those with none of these disorders is not. It is easy to hypothesize momentary changes in coronary circulation with transient ischemia of isolated sections of muscle, a cardiac diathesis which predisposes certain individuals to fibrillation as a result of physiologic stimuli, undetected disease, sensitivity reactions to foreign protein, disturbances af acid-base balance which escape detection and the uncalculated action of ingested substance and many others; these however, receive no experimental substantiation and no real clinical justification. The occurrence of the disorder in such a panoply of conditions which could not be construed as disease states and in conditions in which altered physiology of the body as a whole could not or was not demonstrated, and in the face of demonstrable basis, the comparatively few patients with such disorders as pneumonia, who develop the condition does seem, superficially, to lend some weight to the possibility of a predisposition to the condition, a fibrillation diathesis, as it were, in which the auricular muscle is more sensitive than normal to physiologic stimuli of various sorts.

Clouding immeasurably further conjecture on the occurrence of paroxysmal auricular fibrillation in either normal or diseased states is the paucity of information concerning the actual stimulus to the onset of the attacks. With permanent fibrillation it is consistent with the diseases (or drug states) in which it occurs that it should be present and persistent, but the aberration in cardio-muscular physiology which is instrumental in initiating its rapid onset and equally rapid cessation, the final stimulus to the production of local blocks and selective, isolated prolonged conduction time is not known.

Of those patients with diseased hearts in which paroxysmal auricular fibrillation occurs a definite incidence is seen. In the previously mentioned series of Parkinson and Campbell[10], 15 per cent of their 200 patients had thyrotoxicosis, the remaining 70 per cent (after excluson of the idiopathic group) was divided more or less equally between rheumatic fever, hypertension, and primary myocaridal disease. Of this last group coronary occlusion is the most common. On studying the statistics from the other point of view, it was seen that a large percentage of the patients with mitral stenosis, with thyrotoxicosis and with coronary occlusion have attacks of paroxysmal fibrillation which in many cases becomes permanent. So often do these diseases demonstrate the disorder at some period during their courses that paroxysmal auricular fibrillation is expected and its development guarded against.

The signs and symptoms of paroxysmal auricular fibrillation are as varied as its etiology. In any instance of its occurrence, however, there are certain definite manifestations which may be elicited. With the onset of an attack, the ventricular rate becomes, in the space of one beat, accelerated so that the apex beat lies between 130 and 170 per minute and is grossly irregular. The peripheral pulse very frequently, especially with the more rapid rates, reveals a definite deficit of ten beats or more below that at the apex. The explanation for this is at once simple and obvious. With the onset of fibrillation the auricular rate rises to between 400 and 550 per minute, and this, as has been seen, with great irregularity. The impulses reaching the auriculoventricular node are therefore very rapid and irregular. Because of their rapidity the A-V node and the ventricles are able to respond only to one impulse out of every three or four, thus accounting for the ventricular rate[2], and since these beats are grossly irregular the ventricular response likewise is irregular. There is conceivably another factor in the production of this rate and irregularity. The impulses which reach the node follow along the strands of auricular muscle which converge "like the rays of a fan" upon its auricular end. In a state of fibrillation, with the existence of local blocks and slowed conduction time of some of the muscle fibers, all the impulses do not reach the A-V node simultaneously. The normal discharge of the node is dependent upon the summation of all these individual currents from the separate strands. Thus, the arrival of impulses at the node will, speaking in terms of the summation of all existing individual impulses, vary in intensity. During the absolute refractory period of the node following its discharge, no magnitude of impulse will stimulate it, but once the excitable state is reached the summation of impulses of a voltage greater than its threshhold value at the auricular end may or may not be sufficient once more to excite its tissue. When the necessary summation occurs the impulse is transmitted and the ventricle contracts. The occurrence of an impulse of sufficient magnitude is entirely irregular, and so, thus, is the ventricular rate.

The occurrence of the pulse deficit arises from the transmission by the node of auricular impulse while the ventricles are in a partially refractory state following the previous contraction. This results in a diminished force of contraction which may not be sufficient to raise the aortic valve.

The heart sounds and associated murmurs, if present, are found to vary in intensity from cycle to cycle and, in those contractions which correspond to the pulse deficit, the second heart sound may be lacking. In associated mitral stenosis there is a disappearance of the characteristic presystolic murmur produced by the rhythmic contraction of the auricles.

The symptomatology of the disorder is variable in the extreme, depending to a great extent upon the duration of the paroxysm and the state of the circulation before the attack. In the normal individual, or in he in whom the cardiac reserve is normal, the symptoms are usually minimal, consisting principally of palpitation brought about by the rapidity and gross irregularity of the ventricular rate. In patients whose cardiac reserve has become depleted by the presence of preexisting myocardial disease, the symptoms may become much more severe: dyspnea, which occasionally accompanies the paroxysms in the normal individual, may become pronounced in a short time, in some cases proceeding to acute pulmonary edema with cough and the expectoration of blood-tinged sputum: congestive failure with all its signs and symptoms may become apparent within a few hours in the patient with myocardial disease. An attack of long duration will intensify all the symptoms of failure in these patients as the cardiac reserve becomes progressively diminished. In shorter attacks, even in diseased hearts, there may be no evidence that failure threatened, provided the circulatory condition is such that the disturbance can be countenanced. Conversely, the blood pressure may fall and evidence of shock develop in the person with no other evidence of depletion of myocardial reserve as well as in the cases with frank failure.

In some instances[9] there occur attacks of syncope with the onset of paroxysms of auricular fibrillation; these however, are believed to be rare. In other cases, more common, the onset of the disorder produces varying degrees of typically anginal pain; indeed, Wolff[13] lists two cases in which he feels that a true angina pectoris, in fact a status anginosus, was induced by paroxysmal auricular fibrillation. This is particularly true, according to Foti,[6] if the attack occurs, as it so often does, during the first few days of a coronary occlusion. In some instances it may be the initial pain which the patient has experienced when the occlusion is atypical and in those with few initial manifestations. In this respect paroxysmal attacks of fibrillation may be in some instances indistinguishable from true coronary occlusion on the basis of both pain and the presence of shock, necessitating a most careful examination of the patient and his history in order to institute satisfactory treatment, and to establish the justifiable prognosis.

In rare instances in paroxysmal fibrillation (but much more commonly in the permanent form) and usually only after the paroxysm has existed for several days, does the formation of a clot in the quivering but dynamicaly static auricle occur and with the evidence in many instances of embolic phenomena producing pulmonary infarction with all its signs (if the clot is in the right auricle) or hemiplegias, and other signs of arterial infarction when in the left. When the entire picture of paroxysmal auricular fibrillation is examined, however, embolic phenomena appear to occupy but a very infrequent position, albeit a disastrous one.

In pneumonia, according to Levine,[8] the onset of fibrillation is an extremely serious occurrence, producing complete circulatory collapse.

The bedside diagnosis of paroxysmal fibrillation may, as one may see, be relatively simple or exceedingly difficult. In the instances uncomplicated by anginal pain or cardiac failure, in short in the "normal person" who develops an attack, the diagnosis by physical signs is usually tenable. In those with congestive failure uncomplicated by anginal pain, it is only slightly less so. In the event that substernal pain and shock occur, together or separately, the differential diagnosis between paroxysmal fibrillation and coronary occlusion must be established.

The physical findings of a grossly irregular ventricular beat at the apex whose irregularity is accentuated by exercise, the ventricular rate of 130 to 170 associated with a pulse deficit, the change in the character of the heart sounds and murmurs are all extremely reliable, and if there is also present heart disease, either on an etiologic basis, in the history or detectable by other findings (thyrotoxicosis, mitral stenosis, for example) in which paroxysmal auricular fibrillation is known to occur with some frequency, they may be further strengthened.

In those infrequent instances in which the problems of distinguishing between fibrillation and coronary occlusion arises the most reliable procedure in bedside diagnosis is, according to Foti[6] and to Stubenbord,[12], the history. The subject of paroxysmal auricular fibrillation may have had similar attacks, terminating without sequelae. In acute coronary occlusion a past history of previous attacks would necessarily have been prolonged. However, this is obviously not always reliable for the patient with previous paroxysmal attacks may have coronary occlusion and vice versa. Of greater import than the past history is the different sequence of events in the two conditions: in fibrillation the first symptom is palpitation, in occlusion it is pain. The presence of temperature over 100° and a leukocyte count over 15,000 occurs only in occlusion[6] and on these the diagnosis may be settled. It must be kept in mind however, that fever does not always occur in coronary occlusion.

Electrocardiographic tracings are in all instances immediately diagnostic of paroxysmal auricular fibrillation and serve to distinguish it from any other condition with which it might become confused clinically. The complete absence of the P wave in all leads, the irregular fibrillary deflections present continuously throughout the tracing, and the totally irregular ventricular response with a rate of between 130 and 170 per minute are characteristic of the condition.

The treatment of paroxysmal auricular fibrillation involves two different aspects. One, the treatment of the uderlying condition which is responsible for its occurrence, and secondly the treatment of the fibrillatory attack.

Treatment directed toward the underlying condition is difficult in patients in whom attacks of paroxysmal fibrillation are multiple. Such transient causes as emotional strain, acute febrile illness, for example pneumonia, are difficult only in the sense that they themselves present special problems of treatment. They are not chronically persistent and do not involve irreversible anatomic change which will persist in precipitating the paroxysms. Acute coronary occlusion, likewise, produces fibrillation only during the first few days of its presence and its treatment is, of course, desirable from many aspects other than eliminating the fibrillation. The elimination of thyrotoxicosis by thyroidectomy is not always successful in preventing recurrent attacks of fibrillation (although in many instances it does do so) which, like the exophthalmos, often persists indefinitely. Those conditions associated with anatomic changes in the heart itself, such as mitral stenosis and the permanent mycarditides, are of course, not available for treatment as far as their elimination is concerned. Those people in whom paroxysmal attacks occur without discoverable cause may receive treatment directed toward whatever seems most likely to be responsible; emotional rest, the avoidance of precipitating behavior if such can be determined, may be tried but are likely to meet with little success.

Therapy directed toward the specific disorder, the attack itself, involves both the management of the existing attack and the prevention, by pharmacologic and environmental means, the occurrence of future paroxysms.

The management of the attack of fibrillation encompasses several considerations: the length of the attack, the frequency of separate paroyxsms, the cardiac and circulatory state of the patient and the comfort and psychic reaction of the person to his disorder. In the individual in whom short, infrequent (2 or 3 per year) paroxysms occur without causing much discomfort, it seems generally to be felt that drug therapy need not be employed, the individual being encouraged to bear with them. This has a distinct rational basis. The drugs used in combating fibrillation, both in prevention and in management, are all toxic, and digitalis, if given may serve to entrench the fibrillation. The only management needed, or rather advised in these cases is sedation, preferably by barbiturates, and continued only during the attack and only if the patient becomes anxious and uncomfortable.

When, on the other hand, the attacks occur at frequent intervals, are accompanied by evidences of myocardial failure (dyspnea, increased venous pressure, retarded circulation time) or persist for long intervals—when the fear of permanence is entertained—direct therapy is indicated. Two drugs are employed toward this end, digitalis and quinidine, but with different purposes.

Digitalis acts on the fibrillating heart in four ways: (1) by its stimulation of the vagus it shortens the refractory period and prolongs the conduction time of auricular muscle. These factors together tend to augment fibrillation by increasing the excitable gap in the circus impulse. (2) It acts directly on the auricular muscle lengthening the refractory period and decreasing conduction time; these are essential to the interruption of fibrillation. (3) It acts on the junctional tissue of the A-V node to slow transmission of the impulse to the ventricles, thus producing a ventricular slowing. The first two effects do not reach equilibrium, but usually the first overbalances the second so that the fibrillation tends to be enforced. However, by slowing the ventricular rate through decreasing nodal conduction, the ventricular contraction is made more regular and by (4) increasing the contractility and efficiency of ventricular muscle, they are made stronger. It is for the ventricular effect that digitalis is given. The control of the ventricular rate is desired in any case in which myocardial failure develops or is expected on the basis of preexisting cardiac damage, in which event the actual cessation of the fibrillary movement is of less importance. Besides digitalization of the patient, the rites of rest, and sedation are performed here as in any case of failure.

The use of quinidine (usually as quinidine sulfate), on the other hand, is indicated in those patients in whom the paroxysms occur at frequent intervals, are particularly severe, or continue for long periods of time. Quinidine when used in fibrillation abolishes vagal tone and so lengthens the refractory period of auricular muscle and prolongs the conduction time, it acts directly on the auricular and ventricular muscle to lengthen the refractory period and to slow the transmission rate. It depresses conduction in the junctional tissues. Thus quinidine tends to abolish fibrillation, which it does effectively, and to protect the ventricle from the high and irregular rate of impulse, which it does not always do because of the ability of the node to respond to more of the auricular impulses because of the slowed auricular rate. Although occurring at a slower rate, these impulses are still quite rapid in some instances and may serve to heighten the frequency of ventricular contraction. For this reason the use of quinidine is not favored except in those persons who manifest no circulatory failure. Quinidine is highly toxic, and sensitivity to cinchona alkaloids is a definite contraindication to its use. Marked cardiac damage (as coronary occlusion), a history of embolic phenomena, long standing valvular disease, all speak against its use, and in complete heart block it should not be given for little is gained by attempting to interrupt the attack.

Quinine has been used in the place of quinidine by Horne[5] and earlier by Wenkebach[5] with apparently good results. Its action is that of quinidine, and, according to Horne, its toxic manifestations are much less severe and less likely to occur.

In those patients who require treatment during the attack of paroxysmal auricular fibrillation it is usually desirable to prevent the occurrence of future episodes. This may be done effectively by quinidine sulfate given in doses of 0.3 gram thrice daily. It should be continued for a sufficient time to ascertain whether attacks are being inhibited, if not the drug should be withdrawn. Occassionally, in those instances whe the occurence of an attack is pretictable, the drug may be given before the single paroxysm in order to abort its occurrence.

In fibrillation occurring as a result of coronary occlusion, treatment of the paroxysm is suspended, and directed instead toward the underlying condition.

Paroxysmal auricular fibrillation, as has been stated, is not in itself evidence of myocardial disease, although it frequently occurs as a complication of it, or develops spontaneously more rapidly in patients with some evidence of heart damage. The function of the heart as a pump is invariably impared during attacks of paroxysmal auricular fibrillation, but most often the cardiac reserve is adequate to maintain circulatory equilibrium. Except for those cases in which acute ventricular failure is likely to be precipitated, the great dangers of the disorder are the occurrence of emboli from an intra-auricular thrombus, and the possibility of the fibrillation becoming permanently established as the cardiac mechanism. In these respects the prognosis is satisfactory in those patients with occassional or short episodes, but the increasing frequency of the attacks, and the lengthening of the paroxysms make this danger much more imminent.

SUMMARY

Paroxysmal fibrillation of the auricles is, of all the disorders characterized by paroxysmally rapid heart action, the most benign, and by far the most common. Its intelligent management can do much for the comfort and well being of the patient while offering him, as far as the uncomplicated disorder is concerned, adequate assurance of cardiac health.

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LOUISIANA PHYSICIANS SERVICE, INCORPORATED

In the organization section of the Journal will be found a discussion of the Louisiana Physicians Service, Inc., which is the prepaid medical care plan under the auspices of the State Medical Society. The executive director, Mr. Frank Lais, Jr., has propounded questions which he has answered which will explain in good part the mechanisms whereby the physicians of Louisiana may take advantage of this very forward step in the care of those who participate in the insurance plan. We would urge most wholeheartedly that the doctors of the state back up the Louisiana Physicians Service, Inc. They should be prepared to explain this insurance plan to their participating patients in the lower income group because it is this group which notably has difficulty in meeting their financial obligations when they have a severe or catastrophic illness. They should explain to such patients that the charges are small and that a sum of slightly over ten dollars a year for the individual and slightly over twenty-five dollars for a family will assure medical care for a year.

This plan, worked out by Dr. O. B. Owens and his committee, is one that should have the enthusiastic support of every member of the State Medical Society. It is to be hoped that the members of the organization will do their best to make the plan work notably in order to combat the anticipated by some, socialization of medicine.

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THE HILL-BURTON BILL

This bill has to do with hospital construction and survey and it is an important step in making provisions for hospital service for residents of every state.

The bill, however, does not seem to be of tremendous importance to the doctors of Louisiana. Louisiana pays 48.7 per cent of hospital income from taxes. This is very much higher than any other state in the United States. California ranks next with 37.8 per cent, New Jersey 36.5 whereas in Alabama and New Mexico less than two per cent of the hospital income comes from taxes.

Even more astounding than these figures is the fact that for the support of general hospitals Louisiana pays almost twice as much as any other state. In 1942 for general hospitals Louisiana expended $4,885,000. There was only one other state which paid as much as 50 per cent of the costs of general hospitals; this was Michigan which expended $2,512,000. No other state paid as much as $1,700,000.

Other figures, obtained from a recent publication, "Medical Services by Government," written by B. J. Stern, however, do not put our state in quite such a favorable light. When it comes to the support of mental hospitals, tuberculosis hospitals and institutions for the handicapped, state spending is low. Our expenditure for tuberculosis hospitals is definitely low and for mental hospitals places the state well down the list of all the states. It is obvious from these figures that we have a super-abundance of state supported general hospitals as contrasted with other states. On the other hand we need additional beds for mental hospitals and for tuberculosis institutions as well.

The expenditure for institutions for the handicapped, that is for the blind, the deaf, the mute and the feeble-minded, is about the mean for the states throughout the country.

THE LEUKOCYTIC COUNT IN INFECTIOUS HEPATITIS

Havens and Marck* have had the unique opportunity of observing a series of patients who had experimentally induced infectious hepatitis. These subjects were healthy male human volunteers whose age varied between 19 to 29 years. In addition to their observation on the course of the disease, the symptoms that the patients presented, and the other findings more or less characteristic in infectious hepatitis including jaundice, they made serial observations on the leukocytic response during the incubation period of the disease and its course. It is the response of the white cells to the virus that we wish to comment upon because the leukocytic count is of value in the diagnosis of this interesting disease which has become so frequent during the war period when large groups of young people were collected together.

Twenty-six patients are reported upon in detail. It is to be noted that the leukocytic response begins in the first twenty-four to forty-eight hours of fever and it is associated with the febrile pre-icteric phase of the disease. There develops early a leukopenia, a leukopenia which involves both the neutrophils and the lymphocytes. The charts of these observers show that there is a sharp drop in the total count which may fall as low as 2,000.

The average count is approximately 4,000. With the development of jaundice the count rises gradually to a level between 6,000 and 7,000. The return to normal leukocytic relationship occurs by the end of the second week after the fever has appeared. There are exceptions of course and counts as high as 11,000 may be observed but, as noted before, the average count at time of the fever when the diagnosis is in abeyance because jaundice has not yet appeared, is low.

It should be noted further that there often appears a relative lymphocytosis with numerous atypical lymphocytes. The monocytes are sharply depressed while there is fever. The abnormal lymphocytes producing a relative lymphocytosis are often of the large type so that large lymphocytes may form in as many as 40 to 70 per cent of the total number. A few of these large cells are atypical in appearance, so that their discovery may be of diagnostic importance.

Infectious hepatitis, now known definitely to be a virus disease, is sometimes difficult to recognize before the appearance of jaundice. A series of careful blood counts may be of some help in determining the febrile disease which a patient may have and of which the cause is unknown, prior to jaundice.

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*Havens, Maj. Walter P., Jr. and Marck, Ruth E.: The leukocytic response of patients with experimentally induced infectious hepatitis, Am. J. Med. Sci., 212:129, 1946.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

PREPAID MEDICAL CARE PLAN TO ISSUE POLICY CONTRACTS THIS MONTH

The issuance of contracts by the Louisiana Physicians Service, Inc., the prepaid medical care plan sponsored by the Louisiana State Medical Society, will begin this month, according to Dr. O. B. Owens, president of the new company.

The insurance company was organized as a mutual non-profit association which will provide indemnity coverage to the subscriber for surgical and obstetrical services rendered by a licensed physician.

All doctors in the State of Louisiana are requested to become participating physicians in the plan and are asked to register with the company. There is no fee or cost for registration. During the month letters, pamphlets, physicians service reports as well as the participating physicians agreement will be mailed to each doctor explaining the plan in detail. All doctors are asked to support their prepaid medical care plan as it costs nothing except recognition of the efforts in organizing the plan which it is hoped may be substituted as an alternative to "socialized medicine." Prepaid medical care plans are in operation in nearly every state at this time and have the approval of the American Medical Association. We now ask your cooperation in Louisiana to make this plan the symbol of cooperation between doctors.

The subscriber to the plan will be issued a membership card and the doctor may identify the patient through this card. The doctor should have his secretary contact the branch office indicated on the card to determine if the patient is entitled to the benefits in his contract. The home office in New Orleans or branch office in the hospital service group in the cities of Alexandria, Baton Rouge, Shreveport and Monroe will verify the patient's eligibility for service.

The only other thing the doctor has to do to assure himself that he will receive payment for his service is to have the subscriber sign his name to the assignment contained in the physicians service report, complete the report upon the termination of service and mail the reports monthly to the branch office hospital group in his immediate territory. This system requires less time than billing the patient individually and definitely assures the physician of his fees in accordance with the schedule of benefits if the subscriber is entitled to service.

Participation is voluntary. The participating physician remains free to act in relation to patients in accordance with the custom of ordinary practice. The subscriber to the Society's prepaid medical care plan may select any licensed physician to perform any surgical procedure and the other benefits covered by the contract.

The physician will make his usual charge to patients and the patient will pay that portion of the fee, if any, that remains after payment has been made by the Louisiana Physicians Service, Inc.

For additional information relative to the Society's plan address your communication to the Executive Director, Room 103, 1430 Tulane Avenue, New Orleans 13.

Following is a group of questions and answers which highlight the plan.

What is it?

A non-profit plan for providing surgical and obstetrical care.

Why does it exist?

In order that this care may be made available to all who need it but have previously found it difficult to pay for.

How is payment made easier?

By making small monthly payments into a fund which will provide for the care when it is needed without any additional burden on the subscriber.

Who is sponsoring it?

The Medical Doctors of Louisiana, through the Louisiana State Medical Society.

How will it be sold?

Through the group enrollment plan in use by the non-profit Hospitalization Organizations of the state.

How expensive will it be?

The premiums will be approximately the same as the premiums for group hospitalization; 85 cents per month for subscriber, and $2.10 per month for entire family.

For whom is it intended?

Those employed individuals whose incomes are not sufficient to provide such care for their families without some difficulty.

Can other employed individuals be enrolled?

Yes, under the same conditions of enrollment in groups as the lower income group, and receive the same financial return from the contract.

Where can it be bought?

Through the non-profit hospitalization organizations at Shreveport, Monroe, Alexandria, Baton Rouge, and shortly, we hope, at New Orleans, as well as through the State Home Office.

Where is the State Home Office?

In the City of New Orleans, at 1430 Tulane Ave., where the State Medical Society has its headquarters.

When will it be ready for sale?

Shortly after October 1, 1946, by which time we expect to have a Certificate of authorization to do business from the Insurance Commissioner of Louisiana.

What will it include as benefits?

Payment for surgical and obstetrical care, whether in the hospital or not, and certain related services such as x-ray, anesthesia and laboratory work under certain conditions.

Why doesn't it cover medical cases as well?

The premium necessary to cover such cases would be too high, until the company has established a working reserve sufficient to protect the plan.

Is it planned to expand the services included?

Just as rapidly as is possible. All the profits of the present plan will be used ultimately to expand the coverage as fully as possible, since no person will make a profit out of the operation of the business.

What are some of the things it will do for the physician?

1. Enable him to treat his patients without having to carry the financial responsibility for their care.
2. Make for certainty of collection of accounts for services rendered under the contract.
3. Save the usual costs of collection of accounts.
4. Provide for prompt payment of accounts.
5. Enable him to provide needed services to individuals who formerly delayed as long as possible the receipt of such service because of their financial inability to pay for it.
6. Improve the quality of the physician-patient relationship.

What additional burdens will it place upon the physician?

Practically none.

1. A minimum of clerical work required in making out a statement of the account.
2. If in doubt as to the current eligibility of the subscriber to receive service, check by telephone or otherwise with the local or nearest branch office.
3. Educate your patients in the advantages to them of covering their family need in this simple and convenient way, in advance of their need.

What limitation of choice of physician will be made?

Absolutely none. Any physician licensed as a Doctor of Medicine by the Louisiana State Board of Medical Examiners will be eligible to render services under the contract.

How widespread is this type of protection?

There are similar plans in operation or formation in practically every State in the Union.

Why is this movement so important?

Because American physicians and many other citizens feel that this is the only movement which will adequately meet the need of the American working man for medical care.

What will this plan do to the Wagner-Murray-Dingell type of legislation?

Render it as unnecessary as it is now undesirable and repulsive to the American people.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Oct. 14.	Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
Oct. 15.	I. C. R. R. Hospital Staff, 12 noon Charity Hospital Medical Staff, 8. p. m.
Oct. 16.	Charity Hospital Surgical Staff, 8 p. m. The Orleans Society of X-Ray Technicians, 7:30 p. m.
Oct. 17.	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Oct. 18.	Executive Committee, Hotel Dieu, 8 p. m.
Oct. 21.	Hotel Dieu Staff, 8 p. m.
Oct. 22.	Baptist Hospital Staff, 8 p. m.
Oct. 23.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m. French Hospital Staff, 8 p. m. Catholic Physicians Guild, 8 p. m.
Oct. 24.	DePaul Sanitarium Staff, 8 p. m.
Oct. 25.	L. S. U. Faculty Club, 8 p. m. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Oct. 31.	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Nov. 1.	Ochsner Clinic Staff, 8:30 p. m.
Nov. 4.	Board of Directors, Orleans Parish Medical Society, 8 p. m.
Nov. 5.	Orleans Parish Radiological Society, 7:30 p. m. Eye, Ear, Nose and Throat Hospital, 8 8 p. m.
Nov. 6.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m. Mercy Hospital Staff, 8 p. m.

NEWS ITEMS

Dr. George E. Burch has been awarded a certificate of merit "for original research" by the American Medical Association. The award was made to Dr. Burch for his development of two instruments at the Tulane school of medicine, which are used in the diagnosing of diseases of the blood vessels and heart.

Dr. William B. Clark has just returned from Springfield where he attended the fifth meeting of the Central Illinois Society of Ophthalmology and Otolaryngology, August 3-4.

Dr. Daniel M. Kingsley is temporarily in New York City. He is at the New York Orthopaedic Dispensary and Hospital in order to complete the formal requirements of training for certification in orthopaedic surgery. Dr. Kingsley will remain in New York until July 1, 1947.

Dr. Theo F. Kirn was recently installed as chancellor of the Gentilly Council of the Knights of Columbus.

Dr. J. C. Menendez was recently elected commander of the First District of the American Legion.

Dr. Waldo L. Treuting was recently appointed president of the Louisiana State Board of Health.

CHANGES OF ADDRESS

Dr. George D. B. Berkett from 4900 St. Charles Ave. to 1521 Delachaise St. Dr. Wm. G. Fisher from 1521 Delachaise St. to 1707 Pere Marquette Bldg. Dr. Ira L. Hewitt to 1112 Richards Bldg. Dr. Alfred B. Longacre from 1521 Delachaise St. to 1707 Pere Marquette Bldg. Dr. Charles B. Odom from 1521 Delachaise St. to 1707 Pere Marquette Bldg. Dr. Marie Stanbery from 1206 Hibernia Bldg. to 815 Pere Marquette Bldg. Dr. Walker Thompson from 1430 Tulane Ave. to 4900 St. Charles Ave.

CHANGES IN MEMBERSHIP ROLLS

During the month of July the following changes took place in our membership rolls:

Two associate members (Drs. Jacob Faust and M. F. Wilson) were transferred to inactive membership.

Nine intern members (Drs. Paul L. Getzoff, Lemuel D. Haughton, James C. Jones, Raymond D. Kornegay, Hunter W. May, John J. Milligan, William G. Painter, James F. Stanley, Mary E. Witt) were removed from the rolls because of failure to renew their membership.

WELCOME HOME

The following physicians were recently separated from military service: Michael M. DeBakey, Ochsner Clinic; Charles A. Patterson, Jackson, Mississippi.

NEWS FROM MEMBERS IN MILITARY SERVICE

Lt. (j.g.) Emile A. Bertucci, Jr., MC, USNR, wrote to us from Bikini Atoll on July 4 (aboard the Submarine Tender U. S. S. Fulton AS 11) that he, along with fourteen other doctors, is doing highly confidential and secret work for the bureau of medicine and surgery; they are performing certain studies and autopsies on the animals used in the first test. Lt. Bertucci expects to be separated from the Navy shortly, and will return to New Orleans.

Lt. E. L. Leckert, Jr., MC, USA, is now stationed at Fort Bragg, North Carolina—Regional Hospital No. 2, Post Office Box 43.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LAFOURCHE VALLEY MEDICAL SOCIETY

The third quarterly meeting of this Society was held at the Morgan City High School on Wednesday, September 11.

The scientific portion of the meeting included talks by Drs. P. H. Jones of New Orleans, who spoke of cirrhosis of the liver and Dr. Fred. F. Boyce who spoke on appendicitis.

Following adjournment, a delicious meal was served at the New Shell Inn.

For years our friends, the physicians of St. Mary Parish, have been invited to our meeting. At the meeting in Houma it was voted that they be included in the regular roster as official members of the Society along with all members of the societies of Lafourche, Ascension, Assumption and Terrebonne Parishes.

Our meeting in Morgan City marked the first meeting in this parish.

NEW DEAN OF LOUISIANA STATE UNIVERSITY MEDICAL SCHOOL

Dr. Vernon William Lippard is now functioning as the new dean of the L. S. U. Medical School. Dr. Lippard was formerly Associate Dean at Columbia University prior to which time he was Assistant Dean. He is a pediatrician certified by the American Board of Pediatrics and is a member of the county, state and national medical organizations, as well as belonging to honorary fraternities Alpha Omega Alpha and Sigma Xi. In 1938-39 he was Director of Study, Commission for Study of Crippled Children. During the war he was a Lieutenant Colonel in the U. S. Medical Corps.

Dr. Lippard will be a distinct asset to the medical profession of Louisiana. He has a reputation of being an industrious worker, a man who is interested in the medical profession and one who has a most delightful and agreeable personality.

NEW PROFESSOR OF PEDIATRICS AT L. S. U.

It has been announced that Dr. M. E. Wegman is to succeed the late Dr. Chester Stewart as Professor of Pediatrics at L. S. U. Medical School. Dr. Wegman was Director of the School of Health of the New York City Health Department for four years and at the same time holding the position of Assistant Professor of Clinical Pediatrics and Public Health at Cornell. He has been a lecturer in child hygiene at Johns Hopkins and for eighteen months was Assistant Professor of Child Hygiene at the School of Tropical Medicine in Puerto Rico. He served a residency in pediatrics at Yale Medical School for a period of four years.

TULANE FACULTY PROMOTIONS

The dean of the Tulane Medical School, Dr. Maxwell E. Lapham, announces the following promotions in the School of Medicine, arranged in alphabetical order: Dr. John H. Bethea to instructor in surgery (plastic surgery); Dr. William McDonald Boles to assistant professor of clinical ophthalmology; Dr. Claude G. Callender to instructor in obstetrics and gynecology; Dr. Eugene H. Countiss to assistant professor of obstetrics and gynecology; Dr. Joseph S. D'Antoni to professor of clinical tropical medicine; Dr. Michael E. DeBakey to associate professor of surgery; Dr. Oren R. Depp to instructor in gynecology and obstetrics; Dr. John A. Holmes to assistant professor of obstetrics and gynecology; Dr. Sydney Jacobs to assistant professor of clinical medicine; Dr. Joseph M. Lubitz to associate in pathology; Dr. Mercer G. Lynch to assistant professor of clinical otolaryngology; Dr. John G. Menville to assistant professor of clinical urology; Dr. Leah S. Shaffer to associate in bacteriology; Dr. John R. Snavely to instructor in medicine; Dr. George F. Sustendal to instructor in gynecology and obstetrics; Dr. Roy H. Turner to professor of medicine; Dr. B. Bernard Weinstein to assistant professor of obstetrics and gynecology, and Dr. Jack K. Wickstrom to instructor in orthopedics.

NEWS ITEMS

Dr. W. D. Beacham presented a paper entitled, "The Management of Patients Presenting Large Uterine Myomata" at the fourteenth annual meeting of the Central Association of Obstetricians and Gynecologists held in Chicago September 19-21.

Dr. Guy A. Caldwell, Professor of Clinical Orthopedics, Tulane University School of Medicine, is one of 19 medical men appointed as members of the National Medical and Scientific Advisory Council of the National Arthritis Research Foundation.

Dr. Ambrose Storck, assistant professor of clinical surgery at Tulane University School of Medicine, has been made a consultant in general surgery for the Veterans Administration Hospitals in Louisiana, Mississippi and Texas. He is one of thirteen surgeons who have been appointed as consultants to the Veterans Administration Hospitals.

Dr. George Burch of the Department of Medicine of Tulane Medical School, will give a series of lectures at the University of Minnesota October 2-5. He has also been invited to present a paper before the Inter-American Heart Congress which will be held in Mexico City the following week.

Dr. Michael E. DeBakey has been named one of the members of the new Medical Advisory Committee to the Secretary of War.

Dr. F. L. Jaubert was elected National Historian for the United States and Canada of Phi Rho Sigma Medical Fraternity at its bi-annual convention held recently at Wawase, Indiana.

Dr. William A. Sodeman will speak at the conference of professors of preventive medicine at Ann Arbor, Michigan, September 30-October 4.

At the third annual meeting of the Honorary Consultants to the Army Medical Library held in Washington on October 4-5, Miss Mary Louise Marshall, librarian of Tulane University Medical School, read a paper on the Army Medical Library Classification System.

Miss Vera Morel, medical illustrator of the Tulane Medical School, is one of fifty charter members of the National Association of Medical Illustrators. The first meeting was held in Philadelphia, September 23-26.

The Annual Conference of State Secretaries and Editors will be held at the offices of the American Medical Association in Chicago, Saturday and Sunday, December 7-8, 1946.

The Metropolitan Life Insurance Company is conducting a special campaign on heart disease during the fall and winter months. At that time, the company's more than 20,000 field representatives, in cooperation with official and voluntary agencies, will reach the homes of millions of policyholders with a recently published pamphlet, Your Heart, developed in cooperation with the American Heart Association. A lay educational film on heart disease is also being prepared. Distribution will be made to physicians of a packet in which will be included material of special interest to doctors, and a scientific exhibit on heart disease, first shown at the A.M.A. meeting in San Francisco, is available for State and local professional meetings.

The American Association for the Study of Goiter again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The award will be made at the annual meeting of the Association which will be held in Atlanta, Georgia, April 3-4, 1947, providing essays of sufficient merit are presented in competition.

The Board of Trustees of the United States Pharmacopoeial Convention has authorized the translation of the coming U.S.P. XIII into Spanish, and Dr. A. A. Moll, Assistant Director of the Pan American Sanitary Bureau, will again undertake the translation. Dr. Moll also supervised the translation of both the U.S.P. XI and XII.

United States Pharmacopoeial standards have been widely accepted in Central and South American republics and the U.S.P. has also been looked upon favorably by the Spanish pharmacopoeial commission in Madrid. The demand for the Spanish edition has practically doubled in the past several years, indicating again the closer relationships which are being developed throughout the Americas.

At the Annual Meeting of the American College of Chest Physicians, held at San Francisco, June 27-30, 1946, Dr. Alton Oschner, New Orleans, was elected Governor of the College for the State of Louisiana.

The American Academy of Dermatology and Syphilology will meet in Cleveland, Ohio, December 7-12. The sessions will be held at the Statler Hotel with teaching clinics at Cleveland City Hospital the first three days of the week. It is intended to make this meeting a postgraduate seminar for visiting physicians. Dr. Harold E. Cole is general chairman.

The American Hospital Association has announced the establishment of a department within the association for the purpose of continuing certain of the functions originally initiated by the Commission on Hospital Care which commission disbands on October 1, 1946. The study will be financed by the Kellogg Foundation and the National Foundation for Infantile Paralysis.

COLONEL STEGER HEADS MEDICAL TRAINING CENTER AT CAMP POLK

Establishment of a Medical Training Center at Camp Polk, La., under the jurisdiction of Brooke Army Medical Center, Fort Sam Houston, Texas, was announced recently by Brig. Gen. John M. Willis, Commandant, Brooke Army Medical Center. Camp Polk is located near Leesville, La., southwest of Shreveport.

Commanding officer of the new Center is Col. Byron L. Steger, MC, Gen. Willis announced.

CANCER CONTROL DIVISION OF THE STATE HEALTH DEPARTMENT

Dr. Waldo L. Treuting announces that a Division of Cancer Control has been established in the State Health Department. Dr. James S. May will be the acting head of this division. The funds for operation will be allotted through the United States Public Health Service and will amount to approximately $50,000.00. These funds cannot be expended for treatment or for establishment of cancer clinics, rather they will be employed to make a survey of the state and to promote an educational campaign against cancer.

RESIDENT TRAINING IN SURGERY

Tulane Medical School has just consummated arrangements with Vicksburg Hospital, Inc., to share in a resident training program in surgery. Because of this arrangement Vicksburg Hospital, Inc., has been formally approved for graduate training in surgery by the Council on Medical Education, by the American College of Surgeons and by the American Board of Surgery. The first six months of a residency will be spent in the Department of Surgery in this School of Medicine and the remaining two and one-half years in Vicksburg Hospital, Inc. Dr. Willard H. Parsons is head of the surgical section in this Vicksburg organization.

PACK HEADS MEDICAL ADVISORY COMMITTEE OF NATIONAL CANCER FOUNDATION

Pointing out "one of the most urgent public health problems of the day" is the need of hospital facilities for the treatment and terminal care of the innumerable patients in the United States who have incurable cancer, Dr. George T. Pack, chairman of the medical and scientific advisory council of the recently organized National Cancer Foundation, today (Tuesday, Sept. 10) announced the formation and membership of the Medical Advisory Council. The medical advisory committee of 28 includes Dr. Rudolph Matas.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The next written examination (Part I) for all candidates will be held in various cities of the United States and Canada on Friday, February 7, 1947, at 2:00 p. m. Candidates who successfully complete the Part I examination proceed automatically to the Part II examination held later in the year. All applications must be in the office of the Secretary by November 1, 1946. Candidates in military service are requested to keep the secretary's office closely informed of changes in address.

PHYSICIAN NEEDED

Services of a physician for Merryville, Louisiana, and surrounding territory have been requested by a special committee from that vicinity. It is stated that because of the oil activity in this area many new families have moved into the community and since the nearest physician is 19 miles from Merryville the problem is very acute. Contact the office of the secretary of the State Society if further information is desired in this regard.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending August 3, showed the following reportable diseases in numbers greater than ten: 183 cases of cancer, 139 of unclassified pneumonia, 30 of rheumatic fever, 27 of pulmonary tuberculosis, 24 of pneumococcic pneumonia, 22 of measles, 19 of poliomyelitis, 17 of malaria and 11 of septic sore throat. The poliomyelitis cases were scattered throughout the state, Orleans Parish with three and East Baton Rouge with two, were the only two parishes reporting more than one case. For the week which closed August 10 there were listed 85 cases of malaria, 57 of pulmonary tuberculosis, 24 of septic sore throat, 17 each of unclassified pneumonia and poliomyelitis and ten of typhus fever. Poliomyelitis cases were reported four each from Caddo and East Baton Rouge Parishes, two each from Franklin and Natchitoches and individual cases from other parishes throughout the state. Practically every one of the malaria cases were contracted within continental United States. For the week which closed August 17 there was a sharp decrease in the number of reportable diseases; only three occurring in numbers greater than ten, namely pulmonary tuberculosis with 29 instances, poliomyelitis with 22 and typhus fever with 24. The poliomyelitis cases were again scattered throughout the state, Orleans Parish being the only parish with more than two cases. The typhus fever cases were reported largely from Orleans Parish, there being 14 such instances. The week ending August 24 was also a relatively healthy week except for poliomyelitis, 21 cases of which were reported at this time. The only other two diseases in numbers greater than ten were pulmonary tuberculosis, 44 cases and typhoid fever, 16. The poliomyelitis cases were again diffused with East Baton Rouge Parish being the only parish having more than two cases. The typhoid fever incidence which was rather remarkable, may have been due to a mild epidemic in Bienville Parish where seven cases were listed. For the week ending August 31 the figures were quite different from the previous week, there being recorded 183 cases of cancer, 133 of unclassified pneumonia, 47 of pulmonary tuberculosis, 23 of pneumococcic pneumonia, 17 each of diphtheria and malaria, 16 of poliomyelitis, 14 of measles, 11 of rheumatic fever and 10 of amebiasis. This week East Baton Rouge Parish again had three new cases of poliomyelitis while Caddo and Orleans Parishes reported two each.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending July 31, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	72	338	410
Gonorrhea	1693	10182	11875
Granuloma Inguinale	23	168	191
Lymphopathia Venereum	11	67	78
Syphilis	1908	10053	11961

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending August 10 there occurred in the City of New Orleans 136 deaths, divided 93 white and 43 colored with 20 of these deaths occurring in children under one year of age. The following week the figures were almost identical; two more deaths occurred in the city, three more in the white race and one less in the colored race. The death rate in children was almost exactly the same. For the week which closed August 24, there was a diminution in the death rate in the city, 123 people expired in New Orleans this particular week, 84 of whom were white, 39 non-white and only 12 were in infants. There was a further reduction in the number of deaths in the city in the last week in August when 115 deaths were recorded. Eighty of these deaths were in the white population, 35 in the colored and ten in children under one year of age. In spite of the increase in population in the city the number of deaths recorded each week shows a decided drop.

BOOK REVIEWS

Poet Physicians: An Anthology of Medical Poetry written by physicians: Compiled by Mary Lou McDonough, Springfield, Ill., Chas C. Thomas, 1945. Pp. 210. Price $5.00.

This is an anthology of medical poetry written by physicians and contains examples of poetic endeavors of many physicians who were better physicians than poets and many poets who are relatively poor physicians or merely classified as physicians since they obtained a medical degree, but never truly practiced medicine in the usual sense of the term. The number of true poet physicians who achieved fame in both fields is extremely small. Miss McDonough lists only Weir Mitchell and Oliver Wendell Holmes in this category. Probably a few more might be correctly added to this list.

This book makes interesting reading despite the fact that one might quarrel in many instances with the selection of poems representing various men and also the omission of certain men from her list. However, everything being considered, this forms a very nice collection which is of considerable interest to those who never cease to marvel at the versatility of medical men. An idea of the extensive contribution of medical men to poetry can be more correctly gathered from Danna's bibliography of medical poetry which was published some years ago and the innumerable collections which have occurred from time to time since. This book makes an interesting addition to the medical literary library.

B. B. Weinstein, M. D.

Fractures, Dislocations, and Sprains: By John Albert Key, B. S., M. D., and H. Earle Conwell,

M. D., F. A. C. S., 4th Ed., St. Louis, C. V. Mosby Co., 1946. Illus., pp. 1322. Price, $12.50.

The revised edition of this volume is a welcome addition to the list of reference works on fractures, for surgeons, teachers, and students alike.

The volume is profusely illustrated by x-ray, drawings, and photographs.

There are several chapters which warrant special mention.

In chapter one, devoted to general considerations, the authors have gone to much pains to detail necessary fracture equipment and plaster of paris technic. This chapter should prove of interest especially to those interested in the proper equipment of institutions for the care of these cases.

The chapter devoted to the Workmen's Compensation Law and also the one devoted to Medico-Legal aspects of fracture cases should be read with interest and profit by all interested in the subject of fractures.

The chapters devoted to particular fractures emphasize a phase in the management of the injured which is all too often overlooked, the anatomic considerations involved and the importance of a knowledge of the forces which produce deformity.

The authors are to be congratulated in once again stressing the importance of a carefully conducted clinical examination prior to x-ray studies; such teaching is certainly welcomed at a time when so many are willing to resort to, and depend upon x-ray alone.

Like every subject there are controversial points. One cannot expect to find in the work of others entire agreement with his own views. This volume has the merit that it expresses personal views and convictions based on experience. Honest differences of opinion make for progress. It is easy to recommend this volume for careful study by those interested in fractures.

Isidore Cohn, M. D.

Clinical Case-Taking: By George R. Herrmann, M. D., Ph. D., 3rd ed., St. Louis, C. V. Mosby Co., 1945. Pp. 192. Price, $1.75.

A new edition of this most compact and comprehensive "guide for the study of patients" is always a welcome addition to our teaching armamentarium. By this time it is so well known that it is only necessary to call attention to its publication. It is a "must" on every teacher's program and an invaluable aid to physicians who are desirous of improving their ability to take complete histories and to do thorough physical examinations and to develop an intelligent understanding of the laboratory data essential in establishing a diagnosis.

I. L. Robbins, M. D.

The Surgical Technic of Abdominal Operations: By Julius L. Spivack, M. D., LLD. 4th ed. Springfield, Ill. Charles C. Thomas. 1946. Pp. 712, 682 illus. Price, $10.00.

The need and usefulness of Spivack's "The Surgical Technic of Abdominal Operations" is evidenced by the fact that it is now in its fourth edition. The book records the most common and important abdominal surgical procedures, which is accomplished by clear and concise step by step explanations and illustrations. He discusses the gentle handling of living tissues with a consideration of ligatures, sutures, and the different technics of tying knots. The regional anatomy and surgical technics of important operations on the organs and structures contained in the abdominal cavity are discussed in detail.

The indications, the anatomy, the type of incision in operations upon the small and large bowel, stomach, gallbladder, billary tract, spleen, pancreas and various types of herniae are discussed in a well organized, logical sequence. Some of the more common gynecologic procedures are similarly described. Numerous illustrations and detailed explanations serve to clarify many points in technic and surgical anatomy.

The author has added a great deal to the completeness of the book by discussing not only each surgical procedure, but also its historical background and evolution. An excellent bibliography follows each chapter.

The book has a definite field of usefulness for the beginner during his apprenticeship, as the author points out in the preface, and is in no way meant to replace this period of training. The general surgeon will find it valuable as a review of various surgical technics and a very interesting study from a historical standpoint.

W. I. Waters, M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, New York: Progress in Neurology and Psychiatry, by A. E. Spiegel, M. D. Allergy, By Erich Urbach, M. D., and Philip M. Gottlieb, M. D.

John Hopkins Press, Baltimore: Jewish Luminaries in Medical History, Harry Friedenwald, M. D., D. H. L.

Lea & Febiger, Philadelphia: The Normal Encephalogram, by Leo M. Davidoff, M. D. and Cornelius G. Dyke, M. D. X-ray and Radium in the Treatment of Diseases of the Skin, By George M. MacKee, M. D., and Anthony C. Cipollaro, M. D.

W. B. Saunders Company, Philadelphia: A Primer for Diabetic Patients by Russell M. Wilder, M. D., Ph. D., F. A. C. P.

Charles C. Thomas, Publisher, Springfield, Ill.: Early Ambulation and Related Procedures in Surgical Management, by Daniel J. Liethauser, M. D., F. A. C. S., Renal Hypertension, by Eduardo Braun-Menendez, Juan Carlos Fasciolo, Luis F. Leloir, Juan M. Munoz, Alberto C. Taquini and translated by Lewis Dexter, M. D.

WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

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Mrs. Henry W. E. Walther, New Orleans, 1931-32.
Mrs. Robert T. Lucas, Shreveport, 1932-33.
Mrs. John H. Musser, New Orleans, 1933-34.
Mrs. T. H. Watkins, Lake Charles, 1934-35.
Mrs. Herman B. Gessner, New Orleans, 1935-36.
Mrs. James B. Vaughn, Monroe, 1936-37.
Mrs. George D. Feldner, New Orleans, 1937-38.
Mrs. Frederick G. Ellis, Shreveport, 1938-39.
Mrs. S. M. Blackshear, New Orleans, 1939-40.
Mrs. Roy Carl Young, Covington, 1940-41.
Mrs. Aynaud Hebert, New Orleans, 1941-42.
Mrs. Clarence B. Erickson, Shreveport, 1942-43.
Mrs. George J. Taquino, New Orleans, 1942-43.
Mrs. Rhodes Spedale, Plaquemine, 1944-45.
Mrs. Paul G. Lacroix, New Orleans, 1945-46.

1946 - 1947

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Archives: Mrs. John S. Herring, 1229 Cadiz St., New Orleans.
Bulletin: Mrs. Felix Boizelle, 2255 Kleinert Ave., Baton Rouge.
Cancer Control: Mrs. Clarence Erickson, 423 Herndon Ave., Shreveport.
Commemoration Fund: Mrs. Aynaud Hebert, 2013 Napoleon Ave., New Orleans.
Doctors' Day: Mrs. H. Aubrey White, 2613 Vance Ave., Alexandria.
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Press & Publicity: Mrs. Marcus C. Wiginton, Hammond.
Printing: Mrs. Roy B. Harrison, 2327 Napoleon Ave., New Orleans.
Program: Mrs. Richard T. Stephenson, 2123 Perkins Rd., Baton Rouge.
Public Relations: Mrs. Rhodes Spedale, 701 Eden St., Plaquemine.
Red Cross: Mrs. DeWitt T. Milam, 1704 Island Dr., Monroe.
Revision of By-Laws: Mrs. C. Grenes Cole, 4938 St. Charles Ave., New Orleans.
Year Book: Mrs. Daniel J. Murphy, 127 S. Solomon St., New Orleans.

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MESSAGE OF PAST PRESIDENT OF LOUISIANA STATE MEDICAL SOCIETY TO WOMAN'S AUXILIARY

As president of the State Medical Society, I had the unique distinction and honor of serving with two presidents of the Auxiliary. While the Auxiliary was able to hold its meeting during the year 1945 and install new officers, it was not possible for the State Society to meet and install its officers. Our constitution permits only the House of Delegates to elect and install the officers and since our House of Delegates consists of more than fifty members we could not get permission to hold a meeting in 1945, hence it became my distinction to hold office for two years.

During this period of service the relationship between the Woman's Auxiliary under the leadership of Mrs. Rhodes Spedale in 1945 and Mrs. Paul Lacroix in 1946 was most cordial and the organization never failed to cooperate and give every assistance when called upon. There are many ways in which the Auxiliary can be of benefit to medicine. One of the objects of your organization is "to extend the aims of the Medical Profession through the women members of families of physicians to other organizations which look forward to advancement in Health and Education." The Auxiliary should make it one of its foremost projects — the furnishing of speakers to women's organizations to keep them informed of the aims and purposes of medicine. Also to keep the public informed of adverse legislation that may be introduced both in our own state and nationally. When the public is informed and thoroughly cognizant of just what medical legislation means, they will frequently and almost universally agree with us and see the reasons for our fight against certains laws and regulations. On the other hand when they are not properly informed they are inclined to believe and agree with the politician's point of view. Your organization has had this proved to it on several occasions during the past two years. We, of the State Medical Society, appreciate the efforts put forth by the Auxiliary to accomplish these aims.

Soon the State Medical Society will offer to the residents of Louisiana a prepayment medical service and the Auxiliary can be of a great deal of assistance in acquainting the women's organizations with our plan. The success of prepayment health insurance is one of the best means of combating socialized medicine.

Now that the war is over it will not be necessary for the Auxiliary members to spend time in war work. This time that they so freely gave during the war period can now be utilized to continue to work for medicine.

If, at any time, the Auxiliary needs the advice, counsel or judgment of its parent organization, do not hesitate for a moment to call upon us. I know that our officers are always willing and ready to work and cooperate with you in any worthwhile project.

May the Auxiliary keep up its good work and may it be as fortunate in the selection of its future officers as it has been in the past.

VAL H. FUCHS, M.D., President,
Louisiana State Medical Society.
1944-46

A MESSAGE TO THE WOMAN'S AUXILIARY

As Chairman of the Advisory Board of the Woman's Auxiliary for the years 1944-1946 may I express to you the great satisfaction I feel for the splendid work and cooperation of the Auxiliary with the Louisiana State Medical Society.

There apparently has been very little that has been needed by the Auxiliary from the Advisory Board but you may be sure that any time you might feel that our services may be of value we hope you will not hesitate to call on us.

With felicitations and best wishes for your continued good work, I am

WALDEMAR R. METZ, M.D.,
Chairman, Advisory Council.
1944-46

THE PRESIDENT'S MESSAGE

This is to be my last message to you as your President. What a great privilege to have been so honored!

My years ahead will be richly spiced with lovely memories of ardent, loyal, friendly co-workers who so willingly and courteously responded to every appeal.

Your manifest courage and determination to help our doctors with the legislative problems facing them today has been an outstanding achievement this year.

The reports of our parish presidents and our chairmen indicate how staunchly and actively you have participated in the fields of public health, public welfare and in the vital field of Christian charity and brotherly love.

To expand and improve upon these splendid accomplishments should be our goal.

It would be gratifying to me to know of some especially impressive way to tell you how deeply I have appreciated your help and cooperation; how much I have enjoyed working with you.

Again let me thank you.

I wish to thank Dr. Val Fuchs, President of the Louisiana State Medical Society; the Advisory Committee, Dr. Waldemar Metz, Dr. King Rand and Dr. Rhodes Spedale; Dr. Paul T. Talbot, Secretary-Treasurer of the Louisiana State Medical Society and Dr. John Musser, Editor of the New Orleans Medical and Surgical Journal for their kindly assistance during my term of office.

MRS. PAUL G. LACROIX,
President 1945-46.

IN MEMORIAM

1944	
Mrs. J. T. Prosser	Morgan City
Mrs. A. G. Friedrichs	New Orleans
Mrs. Davidson K. Texada	Alexandria
1945	
Mrs. H. D. Bulloch	Covington
Mrs. Joseph P. Palermo	New Orleans
1946	
Mrs. H. S. Cocram	New Orleans
Mrs. David Jamison	New Orleans
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1946-1947

President: Mrs. Jesse D. Hamer, Phoenix, Arizona.

WOMAN'S AUXILIARY TO SOUTHERN MEDICAL ASSOCIATION

1946-1947

President: Mrs. W. W. Potter, Concord, Tennessee.

President-Elect: Mrs. Wiley R. Buffington, New Orleans, Louisiana.

PROJECTS

1946-1947

Extension of Medical Services Through
Voluntary Prepayment Plans
Periodic Health Examinations
Cancer Control
Red Cross
Hygeia
Anti-Tuberculosis
Doctors' Day
Organization
Commemoration Fund

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Garrett, Mrs. George G., Shreveport.
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Gilmer, Mrs. P. R., Shreveport.
Glass, Mrs. T. A., Shreveport.
Gorton, Mrs. J. M., Shreveport.
Gorton, Mrs. L. W., Shreveport.
Gowen, Mrs. C. R., Shreveport.
Gray, Mrs. Leon F., Shreveport.
Hall, Mrs. W. M., Shreveport.
Hargrove, Mrs. M. D., Shreveport.
Harmon, Mrs. W. S., Shreveport.
Harwell, Mrs. W. R., Shreveport.
Hawkins, Mrs. I. F., Shreveport.
Heard, Mrs. J. E., Shreveport.

Heath, Mrs. A. G., Shreveport.
Heidorn, Mrs. W. B., Shreveport.
Hendrick, Mrs. J. V., Shreveport.
Herold, Mrs. A. A., Shreveport.
Hill, Mrs. W. J., Jr., Shreveport.
Hilton, Mrs. E. T., Shreveport.
Holt, Mrs. C. S., Shreveport.
Johns, Mrs. Barron, Shreveport.
Jones, Mrs. O. O., Shreveport.
Kerlin, Mrs. D. L., Shreveport.
Kerlin, Mrs. W. S., Shreveport.
Knighton, Mrs. J. E., Shreveport.
Knighton, Mrs. J. E., Jr., Shreveport.
LaRue, Mrs. C. L., Shreveport.
LeDoux, Mrs. M. S., Shreveport.
Lloyd, Mrs. T. P., Shreveport.
Lucas, Mrs. R. T., Shreveport.
Mason, Mrs. L. Keith, Shreveport.
Mathews, Mrs. W. R., Shreveport.
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URETHROPLASTY*

J. R. STAMPER, M. D.
SHREVEPORT, LA.

This paper deals with stricture of the urethra, as well as a break in continuity of the urethra from trauma or inflammatory slough, and also with the treatment of stricture of the urethra, as well as repairing the break in continuity of the urethra.

I graduated from medical school in 1916. At that time external urethrotomy was taught as the treatment for urethral obstruction. Since that time, I have seen very little in the literature about its treatment or about perineal surgery. It is my opinion that teaching of perineal surgery is neglected to a large extent. While it is true that urethral obstruction is relieved by external urethrotomy, this obviously produces more scar tissue and more obstruction, and calls for another urethrotomy in a short time. Several years ago there was a wave of advocating opening the stricture with the electric urethrotome; this of course produced much more scar tissue and only lasted for a short time, so it naturally soon fell into disrepute.

The continuity of the urethra is lost either by scar tissue, slowly but progressively forming a contracture until obliteration is brought about, or the continuity is destroyed by trauma, accidental destruction or, it is quite possible in some cases, it is destroyed by the surgeon dissecting it away without recognition.

Strictures should always be recognized early and dilated over a long period of time to prevent excessive scar tissue forming, but it is so insidious in its formation that it is most frequently overlooked by both patient and doctor until irreparable damage is done to the entire urinary tract.

CASE NO. 1

A picture of urogram will be shown in the presentation. A male, age 59, was seen critically ill with dysuria, uremia, and infected urine. Examination revealed an annular stricture in the mid portion of the urethra. The urethra was well dilated above the stricture, and a marked fibrous bar was found at the bladder neck, marked diverticulosis of the bladder, a dilated right ureter and pelvis with poor function; the left ureter was displaced by a diverticulum. The left kidney was functionless and not demonstrable. All of this was continuously overlooked at previous examinations. This could be avoided if all the physicians that attempted to make a complete examination had available some acorn sounds to pass down the urethra and determine if there is narrowing of the lumen. Since the advent of penicillin and sulfonamides, the number of new cases of urethral stricture in the male will be greatly reduced, but we will always have urethral obstruction and destruction from one cause or another, so it behooves us to keep abreast with the management of such conditions.

As long as the continuity of the urethra is intact, and a filiform sound can be passed, continuous dilatation is the treatment to be instituted and is the only treatment that should be used. An external urethrotomy is contraindicated and is harmful. If there are fistulae and multiple strictures, and it is impossible to get a filiform through, you are then justified in doing an external urethrotomy at the point of the most proximal fistulous opening, then retrograde dilatation will sometimes reestablish continuity in the urethra; this is a process that will have to continue during the lifetime of the patient.

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*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

This paper was illustrated by moving picture films, and without the films the paper is somewhat incomplete, but gives the reader to some extent the impression of what the possibilities are in perineal surgery.

If there is a fistula through the perineum and the urine passes through the fistula and the urethra is intact from there outward, the procedure of choice is dissecting the fistula down to the stricture, removing all the scar area and then freeing up both the proximal and distal stumps of the urethra until there is enough free and loose pliable urethra to be brought together and anastomosed end to end. A good deep guy suture should be placed in good tissue above and below and on either side of the repair to prevent any tension appearing at the point of anastomosis. (This will be illustrated in the presentation). This is the most common stricture where there is a complete break in the continuity of the urethra, and is the most satisfactory one to repair as it requires less dilatation, if any, in the future.

CASE NO. 2

A veterinarian, 60 years of age, had been passing most of the urine through the perineum for quite some time, but now having difficulty he applies for relief. A repair by dissection, and end-to-end anastomosis gave him complete comfort. He has been dilated about once annually since that time. This is by far the best type of treatment in the individual who yet has his sexual power.

There is another type of stricture in the person past middle age who has a destruction of the membranous urethra down to the prostatic portion and outward to within the scrotum, also in a person who has a long penis (making more urethra available). This case may have all the urethral bed dissected free of infected tissue and all of the scar removed. Then the corpus is dissected from its attachment on the rami of the pubes and pulled backward through the skin and soft tissue, dissecting the urethra away from the shaft of the penis. As much of the shaft of the penis is sacrificed as is necessary to span the good urethra along the perineum and to the prostatic portion for anastomosis. The stump of the penis is bifurcated and re-attached to the rami of the pubes at its original location. Dorsal penis artery and vein are sacrificed, as well as the sexual power. However, this gives good functional results and requires very little dilatation in the future.

CASE NO. 3

This patient was a male, aged 45 years, with gangrenous posterior urethra from stricture and early extravasation. The perineum was opened, all the urethra dissected from the prostatic portion well into the scrotum, the base of the penis dissected from the rami of the pubes, the artery and vein were ligated. The penis was pulled backward, urethra detached from the shaft and enough of the shaft of the penis was sacrificed to enable the urethra to extend back to the prostatic portion. The shaft of the penis was bifurcated and reattached to its original location, and the urethral stump anastomosed to the prostatic urethra. Urine was drained by an indwelling catheter, and wound closed in the usual manner. Collateral circulation was re-established and the penis lived as well as the urethral attachment. This has been about eight years and the individual has required very little dilatation. He has no sexual power, but has a healthy urinary tract.

There is another type of break in the continuity from either stricture or trauma found in the young individual or in one who is quite active and who does not have a lengthy urethra. This type may be repaired by the skin tube graft taken from the axilla. This procedure will be shown by a moving film in this presentation, also a picture of a case to show the end results of skin tube transplantation.

CASE NO. 4

A boy, 13 years of age, was crushed in an automobile accident, rupturing the posterior urethra. He was carried to a hospital and had a suprapubic cystotomy. A few days later an external urethrotomy was performed with a good deal of difficulty. Evidently the urethra was not clearly recognized, but an indwelling catheter was left in for drainage, and the supra-pubic tube was removed. Healing took place and the catheter was removed from the urethra. He voided urine through the urethra for a few days, then obstructed again. At this time, the urine broke into the rectum, forming a recto-urethral fistula. Atresia of the fistula took place gradually, then it was difficult to pass the urine inot the rectum. At this time some other procedure had to be done. I was asked to see the patient, and selected a skin tube transplantation, which was carried out according to the following technic.

The perineum was opened and dissected to good urethra anteriorly, and scar tissue removed posteriorly to the fistula. This was carried down to the prostatic urethra, which was isolated and freed up, and the fistula dissected back to the rectal wall. At this point, the anal sphincter was severed, the mucous membrane was brought forward and closed, and the sphincter approximated and repaired. The perineum was built up between the rectal wall and the new urethral bed, a skin tube was taken from the axilla and transferred to the urethral bed. A catheter was placed through the penis and tube, into the bladder. One end of the tube was anastomosed to the prostatic urethra and the distal end was anastomosed to the urethral stump. All of this tissue was bathed with penicillin solution. The wound was closed over the tube lightly to prevent pressure and a retention catheter was left in for three weeks. At this time, the catheter came out and he voided well, but it was placed back at each two-day interval, and at one of the procedures, the catheter was pushed back into the rectum and this re-established the recto-urethral fistula. There was so much infiltration of tissue at this area, it was thought best to side-track the fecal matter by colostomy. At the present time, he is wearing a urethral catheter, and efforts to close the fistula are being made.

In conclusion, I might say we physicians owe a great deal to the advent of penicillin and sulfonamides, which enable us to do this type of work and get primary healing.

CALCAREOUS DISEASES OF THE PANCREAS*

MURREL H. KAPLAN, M.D.
NEW ORLEANS

About one year or more ago, while I was serving with a General Hospital in Italy, B. Y., a white male 38 years of age was admitted to my ward with the chief complaint of chronic diarrhea. He dated his illness back to 1938, at which time he was suddenly seized with a severe, excruciating pain in the upper right quadrant of his abdomen. Nausea and vomiting were marked. He was seen by his physician who found his abdomen rigid and who made a diagnosis of possible appendicitis. Large and frequent doses of opiates were necessary for relief. Within four or five days, however, he had sufficiently recovered to undergo a gastrointestinal series. The findings were negative. The diagnosis was made of "colon trouble".

Following this attack, he developed an intermittent diarrhea. This was aggravated by dietary indiscretions and alcoholic indulgences. His stools were frequent, containing some mucous but no blood. Occasionally pain in the upper abdomen was experienced, but it was always relieved by a few doses of codeine.

When I saw this man, he had served in the Army for four years. In the States, he had very little trouble except that he had noticed some digestive disturbance after heavy and greasy meals. Usually he took no medication, but at other times he would resort to a few doses of paregoric. For 24 months this soldier had been on foreign soil. After landing in Africa, he, along with 60,000 or more comrades, had a few "spells" of diarrhea, but he, unlike many of the others, was able to control it with his own medication.

During the ensuing year and a half, he had fought in many battles, receiving three wounds, and had been hospitalized a fourth time for malaria. He was admittedly fatigued by December 1944, when his diarrhea became very intense. He passed between five and 20 bowel movements a day. They were light yellow to dark brown in color, always soft to mushy in consistency and contained varying amounts of mucous. He had lost some 15 pounds in two or three months and was complaining of weakness, anorexia and excessive nervousness when I first saw him.

His past history was significant in that he admitted drinking heavily at times. This began at age 20, but he vehemently denied any chronicity.

The family history was essentially negative except that his mother died at 51 of an unknown stomach ailment. His father died at 63 of kidney trouble. He had five brothers and two sisters living and well. There was no history of diabetes or other metabolic disturbances

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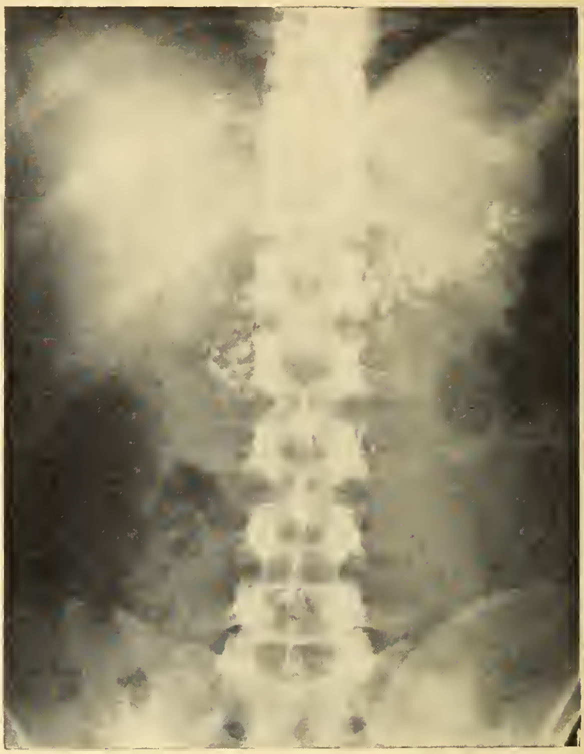
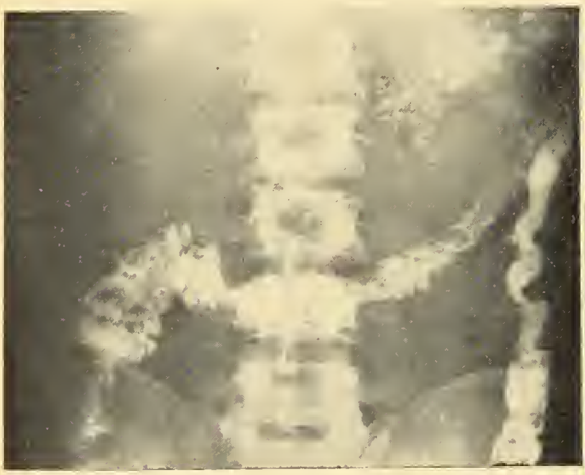
*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.



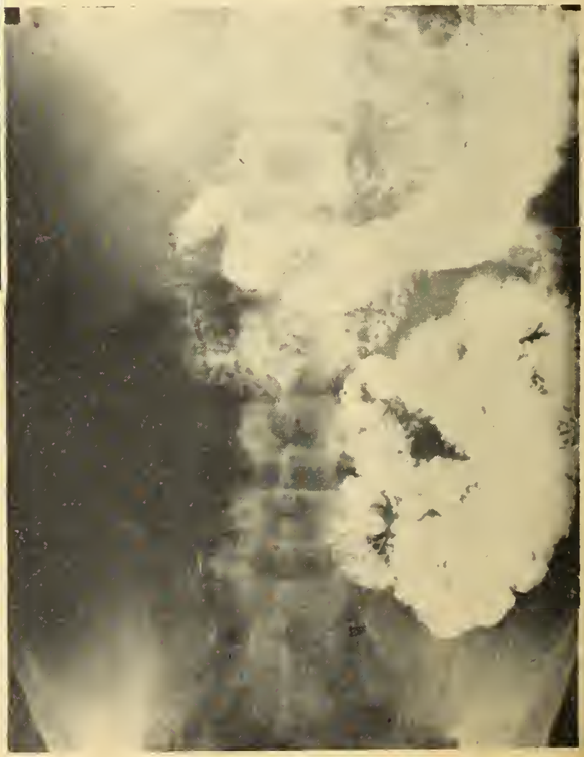
The physical examination revealed a well developed and fairly well nourished male adult, not acutely ill. There was no evidence of any great loss of weight. There was some slight fullness and tenderness in the upper right quadrant, but otherwise there were no positive findings. Routine proctoscopic examination revealed a normal mucous membrane. There was a slight excess of mucous present.

Fecal examinations were negative for ova and parasites, and the cultures were negative for pathogens. On regular diet there was reported, "very much undigested food, moderate amount of fat, neutral fats and fatty acid crystals."



A barium enema was performed with the report negative so far as the colon was concerned, but "numerous small discrete, dense shadows probably calcification, were noted throughout the whole of the pancreas." (Slide 1.) A subsequent flat plate more definitely identified the calcification and confirmed the original impression. (Slide 2.)

Frankly, I had never seen a picture such as this. A gastrointestinal series was then taken further to demonstrate the relationship of this group of calcifications to the stomach and duodenum. (Slide 3.) This, finally and conclusively, was a case of disseminated calcification of the pancreas.



There were many questions which immediately entered my mind. Unfortunately, our library facilities were meager and my scramble into the literature left me anything but satisfied. Consequently, with the aid of our biochemistry and x-ray departments, my investigations were begun with the following results.

LABORATORY FINDINGS

Urinalysis—repeatedly negative for sugar, albumin and microscopic elements. Urinary amylase 100 units (normal).

Fecal studies: (1) Ova, and cysts and parasites—negative; (2) bacteriology—negative for pathogens; (3) food content: (a) On regular diet—very much undigested food, moderate amount of fat, neutral fats and fatty acid crystals; (b) on Schmidt diet—(carbohydrate 200 grams, protein 100 grams, fat 100 grams)—no undigested meat fibers; no excess starch granules, no excess fat—quantitative 13.7 per cent (normal 15-25 per cent).

Gastric analysis: free HC1—66°; total acidity— 94°.

Blood studies: (1) Hematology: red blood cells 5,250,000, hemoglobin 95, white blood cells 11,250, neutrophiles 55, lymphocytes 40, monocytes 3, eosinophiles 2.

(2) Chemistry:

		Normal
N. P. N.	33.3 mg	25-35
Chlorides	473 mg.	350-500
Cholesterol	235 mg.	140-250
Amylase	124 units	70-200
Calcium	11.3 mg.	8.5-11.5
Phosphorous	3.8 mg.	3-4.5

(3) Glucose Tolerance Test

Fasting—115 mg.
30 min.—196 mg.
1 hr. —224 mg.
2 hr. —152 mg.
3 hr. —115 mg.

A typical mild diabetic curve. There was no spillage of sugar in the urine.

(4) Serum proteins: Total 6.3 (normal 6.7), albumin 3.8, globulin 2.5, ratio A/G—1.5.

(5) Cephalin Flocculation = ±/ 2÷ (24/48 hrs.) within normal variation.

(6) Bromsulphalein — Liver Function Test (5 mgm. per kilo body weight) 6/0; (30/60 min.) within normal range.

Duodenal Studies: (1) Bile—"A"—"B"—"C".

(a) Normal concentration. Negative for pus, crystals and parasites.
(b) Culture—"A"—Yeast cells on S. S. media "B"—Yeast cells on S. S. media. Gram positive cocci in pairs and chains, identified as a non-hemolytic streptococcus.

(2) Enzymatic
(a) Tryptic activity 33.3 units (normal 25-50)
(b) Lypolytic activity 0.8 units—(normal 0.2-2.0)
(c) Amylolytic activity 1.2-1-4 units—(normal 2.5-5.0)

Roentgenograms

1. Barium enema—colon negative for pathology.
2. G. I. series—stomach and small intestine negative for pathology.
3. Flat plate—numerous small, discrete, dense shadows probably calcifications are noted throughout the whole of the pancreas.
4. Cholecystogram—gallbladder normal in size, contour and position; no calculi.
5. Chest — lung fields are clear. No evidence of metastasis. A large and small metal foreign body is present. Healed fracture of a rib. No calcifications of the aorta.
6. Osseous structure—skull, long bones, hands and feet; no cystic changes are demonstrated.

The patient remained with us for six to eight weeks. Rest and diet were the only treatment we could offer. With this, he gained four pounds. His diarrhea slowed down to two to four movements daily. He never complained of abdominal pain.

Because of the nature of his illness, he was returned to the States. I have been unable to trace him and, reluctantly, cannot offer any follow-up.

With my "bag" full of data, I returned to this country in late 1945 with intentions of furthering my knowledge of calcareous diseases of the pancreas. The results of this are now offered to you.

HISTORY

As far back as 1667, De Graff described pancreatic lithiasis. Allen in 1903 first reported a case of calcification of the pancreas. It was Beling in 1940 who called our attention to the relationship and yet the difference between the two conditions.[10]

INCIDENCE

Until 1938, over a period of 271 years, only 204 cases were reported.[12] In the subsequent five years, the total ran to 232 of which 19 were of the disseminated variety.[6]

Calcareous disease of the pancreas is obviously then an uncommon condition. The diagnosis in the past was usually made at autopsy and occasionally at the operating table. The increased use of the roentgenograms had made its recognition more common. Autopsies in three different hospitals revealed a total of 26 cases in over 70,000 consecutive postmortems[11] (0.04 per cent). In routine roentgen examinations of the abdomen coupled with the gastrointestinal series, calcifications were demonstrated in 0.07 to 0.13 per cent.[2]

This disease is found predominantly in the male. The average age is 40 years, although one case has been reported with the onset at the age of six.

ETIOLOGY

The etiology is not definitely understood. There seems to be no doubt about the fact that pancreatic lithiasis and general pancreatic calcifications are independent pathologic entities, which, however, probably have a common etiologic basis.[13] It is believed that either an inflammatory, infectious or necrotic process in the pancreas is responsible for the alteration of the chemical composition of the pancreatic secretions and the subsequent deposition of calcium in the ducts[4] and parenchyma. The contributing factors seem to be: (1) bile tract and gallbladder disease; (2) squamous metaplasia of the ducts; (3) desquamated, cornified epithelium may serve as a nidus for calcification in the ducts[8]; (4) invasion by the blood stream or lymphatics of infection from adjacent structures; (5) mumps[7]; (6) chronic alcoholism has been found so consistently, that it is considered more than coincidental (45 per cent of cases).[3]

PATHOLOGY

The exact manner and time necessary for the formation of the calcareous deposits are again unknown. Recent work on acute pancreatic necrosis[9] offers a plausible solution. These observers found large quantities of calcium in acute pancreatic lesions and at the same time noted a moderate fall in the serum calcium of these patients, especially between the third and eleventh day. They reported three cases of tetany associated with acute pancreatic necrosis. The theory[8] is proposed that there is a splitting of the neutral fat by the lipase of the pancreatic juices resulting in glycerin, which is absorbed, and fatty acids which combine with the calcium of the blood to form insoluble soaps.

The calcareous depositions are variously described as organic, containing 12.7 per cent lime salts and inorganic with 93 per cent calcium carbonate.[2]

True stones are present in the ducts and ductules, usually multiple, grayish or brownish gray, hard, rough and angular. False stones, that is, parenchymal concretions, are chalky, gravel-like, in large or small collections, and frequently are mixed with yellowish or chocolate colored organic matter.[8]

The consequences of calculi may be serious. The obstruction of the pancreatic duct, if complete, may lead to an acute pancreatic process, but if incomplete the changes are of a different type. Atrophy and progressive fibrosis with more or less destruction of the parenchyma results. The islets of Langerhans are not significantly involved until the disease is far advanced.

SYMPTOMS

In 1920 Pratt maintained that there was no clear cut symptom complex for pancreatic lithiasis. With the passing of 25 years, this statement is still true, even in the most advanced lesions.

The location, size and mobility of the calculi have much to do in molding the clinical picture. Pain, diarrhea, and symptoms of diabetes mellitus are the most common complaints.

Pain may be dull and transitory or severe and colicky. In some it may resemble pylorospasm or gastrospasm of such a nature as to appear to be functional or psychogenic.[4] The colicky type usually cannot be differentiated from biliary colic, but the radiation to the left side of the abdomen and into the left costovertebral angle is highly suggestive of its pancreatic origin.

In about half of the cases there are signs of pancreatic insufficiency, manifested by frequent, bulky, pale stools containing large amounts of fat and numerous undigested meat fibers. This steatorrhea and creatorrhea are not constant even in this 50 per cent.[4] The marked asthenia and inanition associated with advanced insufficiencies are worthy of mention.

The findings of glycosuria and hyperglycemia as evidence of diabetes mellitus are noted in only about 45 to 50 per cent of the cases of calcareous diseases of the pancreas.

Jaundice is not an infrequent symptom. It was found in 27 per cent of the cases subjected to surgery by Haggard and Kirtley. This is probably due to an obstructive phenomenon, although there may be some few resulting from secondary hepato-cellular damage.

PHYSICAL EXAMINATION

There are no physical findings considered to be pathognomonic. Epigastric tenderness associated with varying degrees of guarding may be present. Icterus and hepatomegaly have been observed.

LABORATORY FINDINGS

The use of the pancreatic function tests have not reached the stage of general availability which would make them particularly helpful in the diagnosis. In acute episodes, an elevation of the blood amylase and lipase[14] may be of definite aid. However, in the chronic phases, these tests are of no value.

The urinalysis reveals a positive sugar test in 20 to 34 per cent of the cases. An increase in the urinary amylase, corresponding to the increase of the enzyme in the blood, is found occasionally during episodes of acute pain.

The glucose tolerance test will denote the degree of hyperglycemia and the severity of the diabetes.

Fecal analysis yields in some cases undigested fats and meat fibers.

The study of the duodenal enzymes may show a normal, decrease or complete absence of pancreatic juices.

ROENTGENOLOGIC EXAMINATION

The most dependable sign, characteristic and diagnostic of calcareous disease is found in the roentgenogram. The shadows are usually dense, multiple and grouped and, in the P-A view, may be seen on either or both sides of the vertebral column, corresponding roughly to the axis of the pancreas. Frequently, they are best visualized in an oblique view.

DIAGNOSIS

The diagnosis of most of the cases has been made accidentally during routine roentgenograms. The symptoms of epigastric pain, pancreatic insufficiency and diabetes should make one highly suspicious of a pancreatitis, but it remains for the x-ray department to demonstrate conclusively the calcium deposition.

In the acute colicky phase, the differential diagnosis should include biliary colic, perforated viscus, renal colic, coronary thrombosis, mesenteric thrombosis, intestinal obstruction and tabetic crisis.[14]

When diarrhea is the presenting complaint, one must rule out the various types of chronic colitis, sprue and idiopathic steatorrhea.

TREATMENT

In asymptomatic pancreatic calcification, therapy is unnecessary. However, regular, interval examinations should be done in order to reveal early evidence of pancreatic failure.

Surgery is usually advised to those who have colic. In capable hands, there is relatively little difficulty caused by postoperative pancreatic fistula or by a reactivation of the pre-existing pancreatitis.[4] In the acute attacks, opiates and fluids are given freely. Ephedrine has been advocated to reduce the volume of pancreatic juice.

If the pancreatic damage is extensive, palliative therapy is the only recourse. The treatment proposed by Beazell, Schmidt and Ivy consists of the administration of enteric coated pancreatic tablets, 8 grams with each meal, 4 before and 4 after.[14] This is increased or decreased, depending on the therapeutic response. Bile salts, vitamin B complex as well as the fat soluble vitamins may help. If there is an associated achylia gastrica, dilute hydrochloric acid may reduce the number of bowel movements and aid in digestion. The permanent limitation of fat is the only dietary restriction.[14] When diabetes mellitus enters the picture, insulin is used accordingly. Patients with fatty changes in the liver may be benefited by use of lipocaic.

COMPLICATIONS

The complications of calcareous disease may include the following:

1. Pancreatic — cysts, pseudocysts, abscesses and carcinoma.
2. Liver — fatty changes, hemachromatosis and portal cirrhosis.
3. Stones may perforate into an adjacent organ or peritoneal cavity with secondary hemorrhage.[15]
4. Predisposition to pulmonary disease as tuberculosis, suppuration and gangrene.[8]

COMMENT

For all practical purposes, you have the story of calcareous diseases of the pancreas. However, before closing, I should like to point out a few facts relative to this case report.

The credit for the diagnosis again belongs to the roentgenologist.

It was intensely interesting to study the disturbed physiology in such an extensively damaged organ. On examining the roentgenogram, I immediately wondered just how much function remained. As you have already seen, the tryptic and lipolytic activities were normal. This is consistent with the literature. The question then must arise, why the steatorrhea and creatorrhea? One observer has contended that this is the result of hypermotility of the bowel secondary to a vitamin deficiency state.[1] The roentgenologic evidence of such a change in the small bowel pattern is lacking in this case. The answer must be then not in the quality of the pancreatic juices but in the quantity secreted. This is brought out more emphatically by the fact that the patient was able to digest the Schmidt diet, but was unable to handle the more liberal regular ward food.

The positive culture of non-hemolytic streptococcus in the gallbladder will find much favorable comment among those who designate the bile tract as the original focus for this pancreatic disease. On the other hand, the cholecystogram and microscopic study of the non-surgical biliary drainage reveal no other evidence of pathology and hence lend no support to this view.

The serum proteins, cephalin flocculation and bromsulphalein, would indicate that the liver had escaped injury.

There seems to be ample proof that there is no parathyroid or chronic calcium metabolic disturbance associated with calcareous disease of the pancreas.

The latent diabetes in this case is worth while mentioning, only to say that there is apparently no correlation between the degree of diabetes and the involvement of the pancreas.[4] This is not at all surprising in view of our present knowledge of diabetes.

That this disease is rare[11] remains to be challenged only by those[4] who have found the incidence higher in their experience. However, regardless of our individual feelings in the matter, we all would agree with Korte who said "that the diagnosis of the disease of the pancreas would be far commoner if the physician would remember that his patient had a pancreas."[4]

I gratefully acknowledge the assistance of Dr. Sam Levi of the x-ray department and Dr. I. I. Zipkin of the biochemistry department.

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DISCUSSION

Question—What are the chances of detecting this condition in routine x-ray examination of the gastrointestinal tract

Answer—In answering this question, I should like to call your attention to slide 3. Despite the fact that calcifications of the pancreas are more dense roentgenologically than the bones of the finger and bladder calculi, they are hardly distinguishable from the barium in the gastrointestinal tract. As you see, it would be a very difficult job, even in this extensive calcification, to interpret the densities above the stomach. Unless one was exceedingly sharp, he would pass those up as part of the small bowel pattern. Certainly, when only one stone or a small patch of calcification was present, it most probably would be overlooked. Beling and his co-workers[11] have advocated a flat plate of the abdomen to precede all gastrointestinal roentgenograms whenever calcareous disease of the pancreas is suspected.

Question—Was there a history of previous attacks suggestive of acute pancreatitis?

Answer—Yes, I would say so. The history of the original attack, which required morphine for relief, certainly had every appearance of acute pancreatitis.

Question—Have you any plausible explanation why there should be this type of calcium deposit in the pancreas when equally as severe pathology, or cell damage elsewhere does not result in this type of calcium deposit?

Answer—I do not think that this type of calcium deposition is peculiar to the pancreas. We see large calcifications in other organs, such as blood vessels, muscles and tendons and lymph glands. Why certain tissues respond with calcium deposition and others with fibrosis, I am unable to say. The presence of the fatty acids and formation of calcium soaps may be the answer, but I will leave that phase to the pathologists.

Question—What is the relation of bile tract disturbances to this condition?

Answer—It is interesting in reading through the literature to find that except for calcareous disease of the pancreas, all other pancreatic inflammations and infections have been associated with or are secondary to infections of the bile tract. Calcifications of the pancreas are found predominantly in the male, whereas biliary disease is most common in the female. Only in 30 to 45 per cent of the cases reported, has there been any evidence of biliary disease.

THE FENESTRATION OPERATION FOR OTOSCLEROSIS:*

ITS POSSIBILITIES AND ITS LIMITATIONS

J. V. McLAURIN, M. D.

BATON ROUGE

In the whole range of medicine there are few conditions more absolutely discouraging from the standpoint of curative therapeutics than deafness. It is not a lethal condition, it is true, nor does it physically incapacitate the victim. However, it does disable him, partially or entirely, in his social and economic relationships, and the psychologic effect is profound, more profound, many observers state, than even the reaction to blindness.

THE ANATOMICO-PATHOLOGIC BASIS OF LOSS OF HEARING

Before discussing the therapy of loss of hearing, let me remind you of the anatomy and physiology of the ear. To state it as simply as possible, air-borne sound waves enter through the outer ear and strike against the tympanic membrane. The vibration thus set up in this membrane is communicated to the ossicles, the chain of small bones in the middle ear. The last of this chain, the stapes, plugs the oval window opening into the inner ear. The vibration of the stapes sets in motion the fluid in the inner ear, and sound impulses are thus transmitted to the organ of Corti, from the organ of Corti to the auditory nerve, and from the auditory nerve to the brain for interpretation. The interruption of sound impulses at any point in the chain of transmission will obviously produce impairment of hearing.

Acquired deafness is divided into two categories, conductive deafness and perceptive deafness. Conductive deafness arises from a pathologic process in the middle ear or the external ear which prevents sound waves from reaching the cochlea. Perceptive deafness arises from a pathologic process in the cochlea itself, or in the nerve pathway. In numerous instances the process is dual, though changes in the outer or middle ear or in the auditory nerve may predominate.

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THE PRESENT STATUS OF THERAPY

With the possible exception of the vitamin therapy recently practiced for certain cases of perceptive deafness, it can be stated categorically that no medical treatment ever devised has permanently improved the hearing of a patient whose impairment was on an organic or structural basis. If the hearing is actually or permanently improved after such therapy it can be assumed that the disability was of psychologic origin. In carefully selected cases of this kind, appropriate treatment, particularly the new method of narcosynthesis used with such success in certain Army hospitals, has produced excellent results.

It is well to point out that while the whole Army Program for the Aural Rehabilitation of the Deafened and Hard of Hearing has been an outstanding success, it is literally a rehabilitation program. It implies an acceptance of the *status quo*. The impairment of hearing is admitted and the patient is taught to adjust himself to it; his degree of auditory impairment is studied by scientific methods. He is fitted —very carefully indeed—with the hearing aid best suited to his special difficulty, and he is trained to use it. He is taught lip reading. He is taught to use to the best advantage such hearing as remains to him. He is given vocational guidance and occupational training as necessary. By these and other measures he learns to live with his infirmity and to rise above it, but his hearing, *per se*, does not improve.

In the light of present knowledge, as has been already mentioned, very little indeed can be done for deafness of nerve origin. Within the past decade, however, a great deal has been done by surgical measures for the variety of hearing loss originating in a middle ear disease known as otosclerosis, though that statement, as I shall repeatedly emphasize, justifies no generalizations as to either universality of treatment or uniformity of results.

OTOSCLEROSIS

Otosclerosis, a term applied both to the lesion which causes a special form of deafness and to the resulting deafness itself, is still of unknown etiology. Infection, abnormalities of development, and defects in the vasomotor mechanism are among the most important theories suggested to explain it.[1] There is some degree of logic in each suggestion, but proof for any single theory is not yet conclusive.

Otosclerosis may be defined as an overgrowth of spongy, vascular bone within the labyrinthine capsule. Its mere presence does not imply loss of hearing. It has been found repeatedly on postmortem examination in persons whose hearing was never affected. The location and extension of the process determines whether or not it is associated with loss of hearing. It may occur in any area of the otic capsule, but by far the most common site (so common, in fact, that it may be called the site of predilection) is the inner wall of the middle ear, just anterior to the oval window. From this point of origin the bony process easily extends to involve the stapes and lock its footplate. As it causes first diminution of mobility and later immobilization of the stapes, increasing impairment of hearing is the inevitable result, because sound waves cannot reach the inner ear. The degree of bony pathology, however, and the degree of auditory impairment are frequently not correlated.

Clinical otosclerosis tends to become manifest late in adolescence or early in adult life. It is much more frequent in women than in men, and many observers believe that it is likely to become more marked with successive pregnancies. Its most striking clinical characteristics are its insidious onset and its slow but steady progression. As the patient grows older, it is frequently associated with nerve degeneration, which in late cases may overshadow the otosclerotic process.

THE FENESTRATION OPERATION

Historical Note: To understand the development of the fenestration operation for otosclerosis, which has been described[2] as "undoubtedly the greatest otologic contribution to medical science of the present generation," a brief historical note is necessary.[3] Since the region of the oval window is the site of predilection for otosclerotic changes, it was natural that the attention of otologists seeking a means of relief for deafness originating in these changes should have been focused upon that area. The first surgical procedures, performed late in the eighteenth century, consisted of a simple opening into the mastoid process. In 1876 Kessel created a fistula into the middle ear by extraction of the immobilized stapes. In 1897 Passow opened the promontory near the oval window, and occasional surgeons over the next two decades created fistulas in various other locations. In every instance immediate improvement in hearing seems to have resulted as soon as the fistula was created, but in no instance was the improvement more than transient. Sometimes regression from the previous level of hearing followed the brief period of improvement, and the operations, aside from their unsatisfactory results, were looked upon with disfavor because of their obvious dangers to life.

After 1917 the attack upon the problem became more intensive. In that year Holmgren made a fistula on the upper curve of the anterior vertical canal and at almost the same time Barany made a fistula into the horizontal canal and covered the opening with a pad of fat. In the latter case improvement in hearing lasted for 14 days. In 1920 Holmgren, using magnifying optical instruments, created an opening into the promontory and covered it with mucoperiosteum, again securing transient improvement in hearing. Two years later he utilized the same technic on the horizontal semicircular canal, and while improvement was not very marked in any instance, it was occasionally permanent.

Shortly afterward Sourdille devised a complicated three-stage operation, by means of which he was able to keep the new window in the horizontal semicircular canal open longer than had been previously possible, and Holmgren, encouraged by these results, continued to refine his own technic. By 1936 he was able to report 34 patients operated on with no deaths and no serious complications; improvement in hearing was constant, and in many instances it was marked.

Holmgren's operation was taken up by Lempert,[4, 5] whose original technic has been successively modified until it now consists of two basic steps: (1) the creation of a fenestra into the surgical dome of the external semicircular canal, and (2) the immediate obliteration of the newly created fenestra with a closely fitting but *mobile* stopple fashioned from auricular cartilage, the same material which covers the inner surface of the normally functioning stapedial foot. The operation thus reproduces as far as is mechanically possible the anatomic and physiologic background against which normal hearing is achieved.

A number of important considerations concerning the operation should be emphasized. The new window is located in the surgical dome of the vestibule, which is adjacent to the area in which the natural oval window is located but which is not the site of predilection for otosclerotic changes and in which, so far as the records show, they have never been found. The insertion of the new stopple not only recreates the normal condition of a mobile stapes but also greatly reduces the risk of labyrinthitis. This can never be disregarded in this vital area. The use of dental drills for much of the bone work and the use of optical instruments to provide magnified vision for the surgeon permit delicacy of technic and somewhat reduce the difficulties of the intricate procedure. The use of constant irrigation during the operation assures the removal of all bone particles which, as has been shown experimentally, may serve as foci for new bone growth.

What the fenestration operation amounts to is a by-passing of the bony growth in the natural oval window and the creation of a new window in an adjacent area not likely to be the site of a similar bony change. It is no more curative than is a gastro-enterostomy for an obstructive gastric ulcer, but it is quite as useful and considerably more logical, since under no circumstances could the offending aural process be extirpated. Lempert,[5] whose experience with the fenestration operation is the most extensive in the world, believes that he has accumulated sufficient evidence to advance the theory that stapedial fixation, if left unchecked, eventually causes atrophy and degeneration of the auditory nerve, which becomes irreversible if it is not interrupted. If this theory is correct, the fenestration operation is quite as logical as cataract extraction done before degeneration of the optic nerve occurs. Whether the operation is done early or late, however, the fenestration operation has absolutely no immediate direct effect upon auditory nerve function.

Selection of Cases: The most important consideration in the fenestration operation is the proper selection of cases. The classical picture of the patient with otosclerosis who is likely to be helped by the operation is about as follows:[5]

1. There is a family history of hereditary deafness, and loss of hearing may become manifest quite early in life.

2. Deafness for air-borne sound is slowly or rapidly progressive in degree and extent. It begins first in the lower frequencies but may ultimately involve the higher frequencies. It is usually bilateral, though it does not necessarily appear at the same time in both ears. The patient frequently states that hearing loss is much less marked in a noisy than in a quiet environment.

3. Unilateral or bilateral tinnitus is frequently present and may be mild or severe and continuous or intermittent.

4. Examination often reveals atrophy or extreme translucence of the tympanic membranes, through which the anatomic structures of the middle ear are clearly visible. The mucous membrane lining the promontory of the cochlea is frequently congested and pinkish. The Eustachian tubes are patent.

5. Audiometric testing demonstrates that inflation of the Eustachian tube does not improve the hearing temporarily, though on clinical testing the patient will frequently claim that it does.

6. Audiometric testing also reveals:
   (a) Superiority of hearing for bone-conducted over air-conducted sound, particularly for low frequencies.
   (b) Maintenance of hearing for bone-conducted sound, though hearing for air-conducted sound may have sunk to a low decibel level.

7. The Weber test is lateralized to the worse ear if the hearing for bone-conducted sound is better in that ear. However, if hearing for bone-conducted sound is equal in both ears it is not lateralized.

Since in otosclerosis as in most other medical conditions the classical picture is the exception rather than the rule, it is practical to select patients for the fenestration operation on the following basis:

1. The patient's hearing for normally spoken conversation has dropped to a level at which it is no longer useful or serviceable or practical for everyday social and economic contacts.

2. The tympanic membrane is intact, and there is no evidence of middle ear inflammation.

3. Audiometric testing reveals:
   (a) That hearing for air-borne sound has dropped below 30 decibels in the conversational frequencies.
   (b) Hearing for bone-conducted sound is prolonged over and above hearing by air conduction in all the frequencies in which hearing by air conduction is impaired.

In other words, the fenestration operation for otosclerosis is, to put it very bluntly, a good gamble when impairment of hearing in the conversational frequencies can be demonstrated not to be the result of degeneration of the auditory nerve and when there is no evidence of gross pathology in the tympanum or the middle ear. As a general rule it should be limited to adolescents and young adults, since degeneration of the auditory nerve becomes more and more likely as age advances, and hearing by bone conduction should not drop more than 20 or 25 decibels at any point in the conversational range. It may be, as Lempert[5] suggests, that these indications may be further liberalized in the future, but for the present it would be well for the otolaryngologist with a less wide experience than his to observe them rather carefully, though it is equally important that he should not permit his own faintness of heart to deny the operation to patients whom it might help.

Results: To date, only a limited number of otolaryngologists have performed the fenestration operation often enough to permit statements of results. Lempert,[5] who has done well over a thousand cases, reported in 1945 that of 815 patients so selected that improvement could reasonably have been expected, 571 had been completely rehabilitated and 92 partially rehabilitated; 118 were not improved, and 34 were worse than before operation. Of 309 patients operated on between three and seven years before the date of the report, 187 had been restored to practical, serviceable hearing which had been continuously maintained, while in 37 others improvement was achieved and maintained but did not reach practical levels; 65 showed no improvement and 20 had become worse. Day[2] in 1945 reported 75 cases, in 54 of which operation had been done between six months and three and a half years before the date of the report; 43 patients, 80 per cent, had been restored to practical hearing levels, including three in whom revision of the original fenestra had been necessary because it had closed.

Standards of postoperative evaluation are, of course, extremely important. Lempert[5] regards a cure as complete if improvement in hearing has lasted for at least a year, though as a matter of fact it is rather generally agreed that if improvement is maintained for six months, the chances are that it will continue. Improvement must be of a degree to permit complete rehabilitation for social and economic contacts as a result of restored hearing, which must be improved to approximately the 30-decibel level or better for the conversational frequencies 512, 1024 and 2048. He regards a patient as partially rehabilitated if his hearing is improved over what it was before operation but not to the practical 30-decibel level in the frequencies mentioned. While testing of postoperative hearing must be done by audiometer, and is preferably done by a dispassionate observer, without other contact with the patient, Day's[2] criterion is useful: If hearing improvement does not reach the level for the comprehension of normal conversation, the operation is a failure, and the best that can be said for the result is that the patient's hearing is not as bad as it was before.

SCOPE AND EVALUATION OF THE FENESTRATION OPERATION

Certain facts about the fenestration operation should be made very clear. In the first place, it is a tedious, time-consuming, delicate, difficult procedure. It can be learned only by exacting application and it should not be undertaken by otolaryngologists who will not take the time and effort thus to learn it. As Lempert[4] puts it, every step leads to the next step, and if a single step is done carelessly, the operation is doomed to failure.

In the second place, the operation is by no means free from risk. It is at least as dangerous as any operation done in the mastoid area, and perhaps more dangerous than most, though in the hands of a competent surgeon this risk is reduced to the irreducible minimum. Careful aseptic technic, both at operation and in the postoperative period, has almost eliminated the risk of infection, but it occasionally occurs unexpectedly, just as it does after any other surgical procedure, even though it is done in a clean field, and the resulting damage to the organ of Corti may be disastrous. No surgical procedure, of course, is free from risk, and the endolymphatic labyrinth and the organ of Corti have been described as the most delicate anatomic structures in the human body. Finally, as Lempert[5] points out, the performance of the fenestration operation does not make the patient immune to untoward non-aural accidents during it; he himself lost two patients from coronary thrombosis soon after operation.

Complications in the hands of a competent surgeon are not the rule. The facial nerve, since it is directly visible, is not likely to be injured except by some accident, though a transient facial paralysis is frequent and may last as long as three weeks. Vertigo after operation may be very severe, though it usually clears up almost entirely by the time the patient has left the hospital, that is, within eight to 14 days after operation.

I am among those who believe that the fenestration operation represents the greatest advance in the treatment of deafness which has ever been made, but I have been deliberately unenthusiastic in my presentation of it. I think it extremely important to emphasize its limitations, its dangers and its difficulties if only because of what it has suffered at the hands of some of its advocates. Nothing more unfortunate could possibly have occurred than the publication in the February, 1945, *Readers' Digest*[6] of an article on the operation which had first appeared in *Hygeia* and so had, at least for lay persons, the semi-official blessing of the American Medical Association.

The title of the article, "And The Deaf Shall Hear," sounds well but is completely erroneous. To be accurate, it should have read, "A certain proportion of a certain category of deafened persons have some chance of regaining some hearing." The statements in the course of the article deserve to be characterized as the author herself characterized the first description of the fenestration technic, that they sound "almost too good to be true," which is precisely what they are.

Whether, as the article states, the greater proportion of the estimated 15,000,000 deafened persons in the country have otosclerosis is most decidedly open to doubt. That 98 per cent of the deafness caused by otosclerosis can be cured by the fenestration operation if the nerve is intact is certainly not true. Some observers put the proportion at 10 per cent, though I myself believe that that is too low. That the operation is either painless or free from risk is equally untrue.

Finally, those who are familiar with the possibilities of the fenestration operation as well as with its limitations can only deprecate such facile statements as "Just as cataracts no longer mean sightless eyes (a statement that can be described only as verging on the reckless) so otosclerosis ceases to condemn its victims to a world of silence," and "For the first time in medical history the doctor no longer needs to inform the deaf patient that there is no cure, no hope, but lip reading or a hearing aid." Quite a number of deafened persons will still do much better with lip reading and a hearing aid than with the fenestration operation, and an unfortunately large number of patients with otosclerosis will still continue to live in a world of silence.

Such glowing accounts of the operation do no good. They raise false hopes, which the otologist must dash when he refuses to operate in inappropriate cases and leaves the patient thinking that he has been denied his chance of cure. The operation itself is discredited if the pleadings of the patient lead the surgeon to operate against his better judgment. Patients who see such failures will not apply for aid, even though they might be in the group which could be helped.

The best interests of the deafened patient demand that his hearing impairment be investigated dispassionately, clinically and audiometrically; that it be determined whether his hearing impairment is: (1) of the otosclerotic type, and if it is, (2) that it is within the range likely to be helped by surgery; that he then be told, beyond chance of misunderstanding, the possibilities of relief and the discomforts and risks of the operation, though it may fairly be pointed out that even if his hearing becomes unilaterally worse after operation, that consideration is little more than academic, since it is already not serviceable. In the light of these facts the patient may then be permitted to decide for himself whether or not he wishes to accept the chance of relief which the fenestration operation offers him.

SUMMARY

The development of the fenestration operation has been traced in its essential principles and it has been pointed out that it is applicable only to the otosclerotic variety of deafness and only to a limited number of carefully selected cases within that category. It is emphasized that unwarranted claims of good results and the performance of the operation in inappropriate cases will discredit what has been correctly described as the most important advance in the therapy of deafness which has ever been made.

Note: Since this paper was completed there has come to my attention a report by J. A. Sullivan (Canad. M. A. J., 53:543, 1945) on 410 fenestration operations, with complete social and economic rehabilitation in 246 patients or 60 per cent. The writer emphasized the importance of exactitude in each step of the procedure.

Also published since this paper was written was a report by Shambaugh (J. A. M. A., 130:999, 1946) on 930 fenestration operations. The highest percentage of improvement, maintained for two years or more, was achieved with the nov-ovalis operation; 246 of the 337 patients or 73 per cent who were operated on sufficiently long ago to be included in the follow-up and who returned for audiographic study showed permanent improvement. The paper also includes an extensive experimental study.

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6. Reader's Digest: And the Deaf Shall Hear, February, 1945.

DISCUSSION

Dr. W. A. Wagner (New Orleans): Certainly we have enjoyed this most interesting and timely subject, and certainly the essayist has brought to us a very good introduction of this new operation, fenestration for otosclerosis. I would like to discuss the subject from the standpoint of practical experience. After six and a half years or more of practical experience with this procedure, it strikes me that there are some fundamental conclusions that one should draw from the end results. There is no question of doubt but that the operation has contributed a big advancement in the handling of that specific type of deafness. There is no question of doubt too, that the operation has some fallacies. For example, I have found out from my personal experience that the operation does restore hearing in otosclerotic deafness. It perhaps can restore as much as 35 decibels, in my experience. It has given excellent rehabilitation. Now, granting that fact, the question then is debatable as to whether a patient should subject himself to a fenestration operation or to a hearing aid. I will admit that there are many handicaps in the use of hearing aids which are perhaps gotten rid of with a successful rehabilitation by means of operation, but let us look at the fallacies of operation. Granting that I can restore 35 decibles after the operation, I can not and have not, been able in the past to maintain that 35 decibel improvement. As most of you know, the window will close either by osteogenesis, fibrosis or irritated labyrinthitis as result of procedure.

There are several procedures that have been described to us; the first operation, of the horizontal canal. I went through that. I opened the horizontal canal and my cases closed.

Then the nov-ovalis operation, whereby the window is made at the junction of the ampulla of anterior vertical and horizontal canals or what is known as the surgical dome of the vesicle. That has been the most successful procedure to me, to date.

Another more recent procedure whereby the cartilage of the helix is placed as a stopple in the newly created window. My experience in the stopple operation is more discouraging that results obtained from opening the horizontal canal and I returned to the nov-ovalis procedure.

I think Dr. Shambaugh in Chicago has contributed quite a lot to the technic in maintaining an open window. For example, he uses a procedure he calls the continuous flow or irrigation procedure. In making his window, continues the flow of saline at body temperature over the operative field. I have found, in using continuous irrigation it interferes with my procedure so I use periodic irrigation. Dr. Shambaugh thinks that the continuous irrigation is superior, he may be right; I cannot contradict him there but let's get back to the end results again.

No matter whether you use the original fenestration in the horizontal canal or non-ovalis, or stopple, my experience has been that the majority of the windows close. I recently made a six year report on end results. I think this represents perhaps an unbiased report of my own experience. Out of 17 cases over six years old to date, there are three rehabilitations. Now out of that group all but two were improved, shortly after operation. All of the 11 have lost some of that improved hearing. Only four have lost a greater amount of hearing than the hearing preoperatively.

My conclusions are these: That if we expect to raise our percentage of rehabilitations we must more carefully select our cases. I have found out that in this group and the recent groups final restoration of hearing may be considered after one year—and one year is considered a sufficient time to expect the hearing to be maintained without closure of the window. In those cases whose hearing gain ended over the one year period about 15 debicels was maintained. Now let me reiterate that and qualify. The maintained improvement has been on an average of about 15 decibels, therefore we should try, in my experience—perhaps I should change too—I should attempt to select my cases in the group whose hearing losses are greater than 30 decibels and not to exceed 50 decibels. If I could select all cases up to 50 decibels I think I could show a larger percentage of rehabilitation. In those individuals whose hearing loss exceeds 60 decibels I finally admit I do not believe I can ever rehabilitate those individuals by the present technic of the fenestration operation. I sincerely mean that, unless my technic is changed.

DISEASE AND THE AGED*

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NEW ORLEANS

The era of diseases of the aged is not starting. It has already begun! In Roman times life expectancy at birth was about 23 years. In 1900 in the United States the figure had risen to 47 years, an increase of 24 years in nearly as many centuries. In 1940 it had risen to 63 years, a rise of a similar period in only four decades. At present over 80 per cent of newborns will live to 50 years of age, over 60 per cent to 65 years. There are now in the United States about 9,000,000 persons who have attained the age of 65. With present trends this figure will be 22,000,000 by 1980. I am not going to burden you further with statistics which have been presented to you many times before. Suffice it to say we are fast becoming a nation of oldsters.

Much of this change has resulted from the application of preventive measures not only to control the environment but also to help the development of increased resistance of individuals to infectious disease. Here, public health procedures have been important. When to these measures are added the use of newer chemotherapeutic and antibiotic agents in treatment, a further reduction in mortality from infections and a rise in the figures for death from degenerative disease can be visualized in the next few decades. The lead gained by diseases of older age groups in our mortality figures in the past forty years, as shown in table 1, is likely to be increased more rapidly than present estimates indicate, because of recent astounding advances in the treatment of infections. Two groups of disease alone, cancer and cardiovascular disease, will probably increase to such an extent that they alone will appreciably affect the total mortality rate. Add to this diabetes mellitus and the rest of the diseases and we can see that disease and the aged is an ever expanding problem which we, the medical profession, cannot meet with the lethargy and neglect which has characterized our approach in the past. A constructive active program is necessary.

TABLE 1

Rank of Leading Causes of Death

1900

1. Tuberculosis, all forms.
2. Pneumonia (probably including influenza).
3. Diseases of the heart.
4. Chronic nephritis.
5. Accidents, all forms.
6. Cerebral hemorrhage and paralysis.
7. Cancer, all forms.
8. Diphtheria.
9. Diarrhea and enteritis.
10. Typhoid fever.

1940

1. Diseases of the heart.
2. Cancer, all forms.
3. Accidents.
4. Cerebral hemorrhage.
5. Tuberculosis, all forms.
6. Nephritis.
7. Pneumonia.
8. Diabetes.
9. Diseases of liver and gallbladder.
10. Arteriosclerosis.

Comparison of the Ten Leading Causes of Death in 1900 and 1940. Note that three groups of infections are no longer in the list in 1940 and two others (tuberculosis and pneumonia) have lost their number one and two positions.

Beginnings have been made in the approach. In 1805 Benjamin Rush discussed the problem. Others, following him, sought means of prolonging the natural span of life. Such were the hopes held out for acidophilus milk and for operative procedures removing several feet of the intestine. But it was J. L. Nascher who introduced the modern concept of the care of the aged. He died in 1944. His first important article appeared in 1908[1], and his textbook entitled *Geriatrics* appeared in 1914. This present year has seen the establishment of the journal, *Geriatrics,* devoted to research and clinical study of the diseases and processes of the aged and aging.

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The problems of aging fall into three categories: (1) the biology of senescence as a process; (2) the clinical problems of senescence, and (3) the socio-economic problems of aging. The first two are always important to the practice of medicine, the third may be especially so in industrial medicine. The process of aging produces many important changes. The desiccation of tissue, slowing of cell division, atrophy, diminution of muscle strength, reduced elasticity of tissues and impaired keenness of the special senses, create special problems of old age. Patients react differently to disease. Therefore, their symptoms may be different. The older body differs so the signs may be different. Changed chemistry may influence laboratory data. Finally, treatment must fit this changed old body. Just as children are not small adults and create special problems for the pediatrician, so the elderly patient differs from the young adult and creates special problems.

Such remarks naturally lead to consideration of geriatrics as a specialty. In my opinion there is little general need for specialization in geriatrics. The term, like the term *psychosomatic,* is important in that it focuses attention on a problem. To be sure, in some large centers and densely populated areas, some may devote their full time to the study and care of the aged, but by and large this must be the problem of general practitioners. It is essential to remember that many of the factors contributing to disease in old age have their beginnings in other years and treatment at that time is most important. What, then, are the problems that confront us and how can they be met?*

The measures applied in the control of the environment, such as those effective against typhoid fever, as well as those effective against such problems as dusts, fumes, and noxious gases in industry, have been eminently successful as exemplified by the changes in mortality statistics already mentioned. Such methods have been applied almost without thought given them by the individual and even the physician in large part. In urban areas good water supply and adequate sewage disposal are accepted by most of us without much thought. The effectiveness of preventive measures in suppressing infectious diseases in the past has rested upon the mass approach made possible through control of a known etiologic agent which should be blocked in the environment. In the diseases affecting the aged, such conditions do not prevail. In malignant disease, metabolic disorders, hypertension and arteriosclerotic damage, to cite a few examples, the etiology is obscure and, to our knowledge, chiefly endogenous. The clinical picture is often insidious in its onset and the course chronic and progressive.[2] Against such diseases, therefore, the preventive measures applicable to infections are ineffective. Preventive procedures, just as therapeutic measures, must be individualized. The subjects cannot be approached in mass but must be examined and studied singly. Prevention must be carried out within the limits of the practice of medicine. Some preventive measures have long been applied to the individual (typhoid vaccine, diphtheria toxoid, and so on), but in large part active preventive programs with individualized approach have been applied extensively in two fields, pediatric and obstetrical practice. As Stieglitz[2] notes, modern pediatric practice has demonstrated convincingly that the health of well babies can be improved, that guidance in nutrition, regulation of exercise, instruction in sane cleanliness, and advice regarding wiser habits of living, do develop healthier children. It is absurd, he states, that such medical guidance should be applied only to infants and children.

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*Geriatric medicine is a part of the larger problem of chronic disease, a field in which facilities for care are woefully lacking, except in tuberculosis and psychiatry. The broad aspects of the problem of chronic disease in general will not be discussed here.

Other factors produce difficulties in prevention and control of disease of the elderly. Because of lack of symptoms, early diagnosis is difficult. Then, too, there is a general low individual interest in prophylaxis. Most important is the lack of drama in preventive treatment. The patient cannot see the benefit as he does in a rapidly moving acute symptomatic picture. However, the attitude of the physician is our most important stumbling block now. Many of us in our investigation of the elderly patient look no further than for some assurance that immediately important changes are absent. If the lungs are clear and the heart compensated, we pat the old man on the back and assure him he is all right. A few of us—perhaps, we feel, more enlightened—may give him some advice as to how to avoid disease. Only too few of us, however, approach the ideal of seeing in the patient, especially when he has little or nothing in the way of complaints, the opportunity to apply the principle not only of prevention of disease but of improvement of health. This is not a new concept but a further application of the principle extensively applied in children for many years.

I have already pointed out the differences in symptoms and signs produced by disease in the old patient as the result of changes of senescence. These, together with the changed physiology producing them, cause too few of us to see in our older patient some of the advances of active disease. For example, in the elderly, emphysema and concomitant bronchial changes producing cough are not uncommon. The same is true of bronchiectasis and heart disease. Such changes make for difficulty in the recognition of other disease developing in the chest. Tuberculosis, which is known to be frequently unrecognized in the elderly, may advance in the chest, produce cough, loss of weight and other symptoms in the presence of a positive sputum and long be overlooked because old patients show little in the way of toxic effects. There are many elderly tuberculous patients in the United States in whom detection of the disease is not simple. If the physician assumes the cough and related symptoms are due to pulmonary and bronchial changes resulting from age, he will never help these patients and will fail to protect against infection those with whom they live.

Nutritional disease is another field in which vague nondescript symptoms are likely to be misinterpreted. Old people, especially if they live alone, are likely to eat a monotonous diet. Their desire for a variety of food sometimes diminishes. The difficulties of traveling to the store and of preparing food for a proper diet often lead to repeated use of the same foodstuffs. The amount of food eaten becomes reduced. I have seen many old people who have lived for weeks chiefly on bread and coffee. If dietary histories are not obtained, the weakness, numbness, incoordination, and lassitude due to subnutrition are very often interpreted as the result of arteriosclerosis or "old age." I have seen remarkable "cures" in such people effected by nothing more than adequate nutritional therapy.

Clearly then one of our problems in the care of the elderly patient is the physician's attitude toward the patient. The patient's symptoms should be given the same consideration and analysis accorded those of the younger group.

Another factor of importance in disease of the aged, again already mentioned, is the physician's lack of interest in diseases prevalent in later life. Syndromes on an arteriosclerotic basis, mild diabetes, hypertensive disease, are not curable. I have often heard the attitude expressed, "You can't do anything for them, why worry greatly about them." This is not true. I do not believe that the day of medical magic will arrive when these disturbances will be eliminated. Indeed, I think they will increase. I do not hope for a cure for the process of aging. I think it is a natural process and that beyond a certain point, we cannot add years to our lives. But I do believe the statement that we can add life to our years. Well controlled insurance statistics show that patients with degenerative diseases who undergo physical examinations frequently and are given advice as to proper care and control of their activities do get along much better and that mortality statistics are improved. Much of the chronic disease present in older age groups, arthritis and hay fever and asthma, for example, is not killing and does not contribute greatly to mortality statistics. The relief that many people with these diseases obtain from proper medical care is tremendous.

A further consideration of importance, implied from remarks already made, is the attitude of the physician on matters of health improvement. The absence of positive physical findings or other evidences to establish a specific diagnosis is *not* the signal to dismiss the patient. His habits of eating and exercise, his rules of bodily care, factors of clothing, housing, and daily routine should be reviewed to find the points at which his health may be improved or where beginning deterioration may be blocked. This concept makes the difference between the modern approach to disease and the medicine of the Middle Ages.

Finally, I must stress again a point of importance in the care of the elderly. This is the recognition by the physician of certain peculiarities which age gives to disease. This is well known in pneumonia, for example, where the febrile response and the general symptoms do not find the dramatic expression they do in the younger group. Complications, too, are often different. I not uncommonly see, in the elderly patient hospitalized for cardiac and pulmonary disease, the development of disorientation, irritability, and emotional instability coming on rapidly and progressing until there is a clouding of consciousness or even coma. The patient may develop fears at night or have his symptoms only at that time. Muscular incoordination, rigidity of muscles and tremors may appear. Recovery usually follows proper supportive and symptomatic care. These symptoms rest upon cerebrovascular changes as a result of bed rest in the elderly. They do not occur in youth. They are a part of the problem of the treatment of disease in senescence.

The points discussed above show the benefits to be derived from the proper care of the elderly patient as an individual. Even though the numbers of our population above the age of fifty increase to the extent that, from numbers alone, the problem is a public health one, the mass approach applied by public health departments to the control and eradication of such diseases as typhoid fever and even tuberculosis cannot be applied here. The nature of the diseases is different. Sadly enough, the etiology of many of the disturbances is unknown. If the causes were discovered and if means of control were established, the approach would be radically different. With our present knowledge an attack on the environment would not seem a likely approach. Individual care would be necessary for each individual patient. The technics of the cancer detection and prenatal clinic would probably be necessary, a program which, with proper education of the public and the proper attitude of the physician, could be as well—likely even better—carried out in the office of the private physician. In geriatrics, as in pediatrics, adequate clinical facilities in the hands of an interested and alert clinician will give the best results in our present state of medical knowledge. Here, preventive procedures are individual clinical problems best cared for in the office of the practicing physician. Only the alertness and adequacy of the medical profession in solving this problem in the rapidly expanding field of geriatric medicine will settle the need for health department sponsorship of health examinations and community provision for such facilities. We have delayed too long in attacking this problem.

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DISCUSSION

Dr. E. L. Wenk (Cloutierville): There are at present two geriatric journals being published, *Geriatrics*, official journal of the American Geriatric Society, and *Journal of Gerontology*, published by Charles C. Thomas of Springfield, Ohio.

I think Dr. Sodeman has made an excellent presentation. However, I would like to re-emphasize the use of the term annual health inventory and not health examination; the objective of the annual health inventory being to detect, by means of stress fuction tests, early degenerative processes which may not otherwise be discovered.

Dr. J. E. Knighton (Shreveport): I do not think I can add anything to the information that the doctor brought us; he has given a most excellent discussion of the subject. I am often reminded of a remark that I have heard my past associate, Dr. Willis, Sr., make on many occasions—that if we continue to live we are going to grow older, and that he did not know but one way to keep from getting old and that was to die young and not many people would voluntarily select that means to avoid old age.

With reference to how people should take care of themselves as they advance in years, I think that we should remember that as we grow older it is a natural course of events for us not to have resistance and resiliency as in earlier life and just to look at the problem in a common sense way. Only last evening a doctor friend of mine here in attendance at the meeting—he is 67 years old I believe he said—told me that when he walked from his office to the hospital there was a drug store about three blocks away where he had to rest because he felt pain in his chest. He wanted to know what I thought about it. I said that means that you are developing some inflammation with reference to yourself that you ought to heed. If a man cannot walk more than three blocks without developing pain in his chest I think every doctor knows that he should not abuse that warning. This is not only true in that particular thing but there are many other danger signals which we should heed as we pass along the pathway of life. A good many of my friends, even here in this meeting, have complimented me by telling me that I do not look older than I did ten years ago. I know they are just joking.

I am still reminded of what Dr. Willis said, "Keep living to grow older."

Question—At what age did the author of the text on geriatrics die?

Answer—About sixty-three or sixty-four.

Dr. W. A. Sodeman (in closing): I am glad some remarks were made about the stress tests in the care of elderly people. That was not a part of the thesis I was to develop so I could not say much about it. The remarks give me an opportunity to add something. Of course as we get older and as the reserve power in various organs in the body become diminished we see a greater importance in determining, by stress tests, the limits of our ability to adapt. Such tests are particularly important not only in that they complete the routine examinations given to individuals from time to time for comparative study but because they point out to you as well that a health examination is not merely a matter of taking a quick history on someone and doing a physical examination to elicit a murmur, or feel a liver, and so on. Health examinations are more than merely an examining procedure to be carried out; they should evaluate the individual as he fits into his environment and show how he reacts to it and how he acts if it is changed. Therefore you have to know something about the individual's habits, background, what he does about it, the extent to which he uses various functions in his body and, if possible, improve those things for him. You must know whether he has reached the limits or has come near to reaching the limits of some of the reserve powers in his body. If you are able to do so and carry out a review in that way, you have really investigated that individual. I do not think there is anything that has put health examinations in more disrepute than the forms as brought out for such procedures by the A.M.A., blanks where you check on a line one or another thing about a patient. Nothing will let you treat a patient in a factory way more than that. A health examination should evaluate the state of health and cannot carry out by that procedure.

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THE PATHOGENESIS OF POSTOPERATIVE ATELECTASIS*

CLIFFORD J. VEDRENNE, M. D.
NEW ORLEANS

The word "atelectasis" is derived from two Greek words, "ateles" meaning imperfect, and "ektasis" meaning expansion. It was first used by Joerg (cited by Adams[1]) in 1832 to describe the lack of inflation of the lungs of newborn infants as seen postmortem. Since then its meaning has been extended to include clinical cases in which there is collapse of lung tissue due to an absence or marked diminution in the amount of air in the alveoli of a whole or of a portion of lung. Jackson[39, 40] described three types of atelectasis according to its distribution:

1. Lobular or partial (also referred to as patchy): The upper or middle third of one or more lobes is the most frequently affected part. There are scattered areas of atelectasis, usually with emphysematous

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areas between them. The most frequent location is the right lower lobe.

2. Lobar: One or more whole lobes are collapsed. The right lower lobe is the most frequent location. Pasteur originated the term "massive collapse of the lung" to describe this type.

3. Total: The whole of one or both lungs is collapsed.

HISTORY

In the early days of anesthesia, the local complications of surgery were so severe and were so frequently encountered that postoperative pulmonary complications were either entirely overlooked, or, when observed postmortem, they were interpreted as terminal conditions. However, with the development of asepsis and improvement in surgical technic, the incidence of local complications markedly diminished, so that the pulmonary complications then attracted attention and became recognized as very important sequelae of surgery. At first, the reason why these latter conditions developed appeared to be self-evident, for it seemed logical to infer that they were caused by the irritating effect of ether vapor upon the lungs. For a long time this action of ether was considered to be the only factor, or, at least, the most important one responsible for the development of these lesions. Of course, the skill of the anesthetist was seriously questioned, and was considered by some to be the most important factor involved. Even today there are those who put the whole blame on the anesthetist. As a result of this theory, all postoperative pulmonary complications were designated as either "ether bronchitis" or "ether pneumonia," depending upon the severity of the symptoms. Later, with the recognition of pulmonary embolism as a postoperative complication, at least part of the blame was diverted from the anesthesia to the surgical technic. Yet, the true picture was destined to remain obscure for a long time, because the condition, which is most frequently responsible for postoperative complications, although it had been recognized clinically as a post-diphtheritic complication by William Pasteur in 1886, was not even considered as a possibility in these cases until Sir James Barr[7] called attention to the occurrence of atelectasis following operation. In 1907 he reported it in a youth a few days following the removal of tuberculous glands from the right iliac fossa. Nevertheless its importance, as a pulmonary complication following abdominal operations, did not become apparent until William Pasteur[60, 61, 62] published a series of articles on the subject between 1908 and 1914. He first suspected, then recognized, that the so-called "postoperative pneumonia" was, in many cases, massive atelectasis rather than pneumonic consolidation. In 1914 Elliott and Dingley[26] corroborated Pasteur's observations and emphasized its clinical importance. They also indicated that it followed spinal as well as inhalation anesthesia. Furthermore, they reasoned that the most likely cause was obstruction of the bronchioles by inflammatory edema and secretion, these secretions blocking the tubes easily on account of lessened ventilating current resulting from weakened diaphragmatic action. In spite of all this, general recognition of its importance and frequency was slow to spread. Likewise, the part played by the various etiologic factors involved in its production, as well as its actual mechanism of production, was even slower to be appreciated. The complete solution of the problem was destined to await the observations and researches of many workers, so that all the factors could be properly evaluated and correlated. The outstanding researches of Coryllos revealed the decisive role played by bronchial obstruction in these cases. Coincidentally, clinical observations by Chevalier Jackson[39] led him to point out the similarity of the symptoms and x-ray pictures in these cases and those of bronchial foreign bodies.

INCIDENCE

Coryllos[18] cautions us to remember that statistics are incomplete on account of the frequent failure to diagnose atelectasis. Notwithstanding the fact that it is the most common postoperative pulmonary complication,[1, 56] it is often overlooked or unrecognized, and is often diagnosed as postoperative pneumonia and reported as such;[40] therefore, much confusion exists in the reported incidence of both conditions. Most reports include them combined in one group. However, this need not perplex us, for, as will be described later, pneumonia is a sequel to atelectasis, so that the whole group is atelectasis in the beginning. At hospitals, where an effort is made to differentiate between these two complications, the incidence of postoperative atelectasis rises while that of postoperative pneumonia falls, and, the greater the effort employed, the greater the incidence of atelectasis becomes.[51] In enumerating these cases, a further source of error probably results from the diagnosis of some cases of atelectasis as pulmonary embolism, on account of the sudden attack of severe pain in the chest.

Most of the cases of both postoperative atelectasis and postoperative pneumonia occur after abdominal operations, especially those in the upper abdomen.

INCIDENCE OF POSTOPERATIVE ATELECTASIS AND PNEUMONIA

	Abdominal Operations Per cent	Other Operations Per cent
King (cited by Adams[1])	14.3	1.2
Rink (cited by Adams[1])	14.0	1.0
Mandl (cited by Elwyn[27])	6.29	0.7

At the Lahey Clinic the incidence of postoperative lung complications was found to be greater after operations on the stomach than after operations on the gallbladder. Their figures, reported by Sise, Mason and Bogan,[76] are as follows:

	Per cent
Stomach operations	14.8
Gallbladder operations	5.9
Average of these two	7.7

Mandl (cited by Elwyn[27]) reported an incidence of 13.8 per cent following operations on the stomach.

Perrone[63] reported 90 cases in which atelectasis most commonly followed operations in the upper abdomen.

Cutler and Hunt (cited by Churchill and McNeil[15]) found the incidence after various operations to be approximately as follows:

	Per cent
Epigastric operations	8
Laparotomies	4
All operations	2

ETIOLOGY AND PATHOGENESIS

Since most of these cases occur after abdominal surgery, the underlying cause must be some factor, or combination of factors, commonly present after laparotomy; furthermore, these factors must be present to a greater degree after upper abdominal than after lower abdominal surgery; and they must be absent, or present to only a slight degree, after other operations. As a matter of fact, the solution of the problem depends upon the correct correlation of a great many factors which form a complex chain of events leading to an end result, that is, atelectasis. However, one factor is conspicuous above all others, that is, bronchial obstruction. As early as 1878, Lichtheim (cited by Elliott and Dingley[26]) produced atelectasis in rabbits by obstructing a bronchus with a laminaria plug. He also demonstrated that collapse did not follow obstruction if the blood vessels to the lung were tied, thus proving that collapse of the alveoli was not an active, but, a passive process, depending upon absorption of their contained air into the blood stream. The more recent extensive experimental work of Coryllos[17] and his associates has demonstrated the importance of complete bronchial obstruction as the one factor that determines the development of atelectasis. All other factors act as predisposing and favoring causes; they cannot produce atelectasis by themselves, although Burford[12] believes that extremely shallow breathing, by not ventilating the peripheral alveoli, can lead to collapse of these with resulting patchy atelectasis, without the presence of bronchial occlusion. However this may be, complete bronchial obstruction stands out as the one condition which definitely leads to atelectasis. Once this occlusion is established, the air in the alveoli, tributary to the occluded passage or passages, becomes absorbed by the blood; and, since no new supply of air can enter, collapse of these alveoli must follow. The type of atelectasis that develops depends upon the size of the obstructed passage or passages.[21] Obstruction of the smaller bronchioles will lead to lobular atelectasis, while obstruction of a lobar bronchus will lead to lobar atelectasis, and obstruction of a main bronchus will lead to collapse of a whole lung.

Coryllos' observations have been verified clinically many times. Jackson[39] was the first to compare these cases with massive atelectasis which complicates foreign bodies in the bronchi. He noticed the similarity of symptoms and x-ray findings in both groups. Tucker,[81] a member of Jackson's clinic, was the first person to demonstrate bronchial obstruction in a clinical case of postoperative atelectasis. With the use of the bronchoscope he found a plug of thick, tenacious mucus causing complete obstruction in the main bronchus and its subdivisions leading to the atelectatic lung. After removal of the plug the lung returned to normal. Since then he and other bronchoscopists have made many similar observations. Thus complete bronchial obstruction is generally accepted as the immediate precursor of atelectasis.

Having arrived at this conclusion, our next step is to determine the manner in which this obstruction occurs. Theoretically, there are three possible ways in which the bronchial tree may become obstructed:

1. Reflex bronchospasm.
2. Swelling of the bronchial mucous membrane.
3. A plug formed by the presence of normal or abnormal secretions or by any aspirated material.

Reflex Bronchospasm: Some observers, notably Scott,[73], Cutler[75], and de Takats,[25] believe that stimuli arising in the field of operation give rise to a nerve reflex, probably through the vagus or sympathetic system, which produces bronchoconstriction. They believe that all other factors, including the tenacious mucus, are secondary. Even Coryllos (cited by Adams[1]) agrees that a small minority of cases may be due to this mechanism. In experiments on dogs, de Takats[25] observed that vagal stimulation, pulmonary embolism, manipulation of intraabdominal organs, and blunt injury to the chest wall all produce bronchoconstriction and increased bronchial secretion, a combination of conditions that favor obstruction. He also demonstrated that this reaction could be inhibited either by section of both vagus nerves or by the intravenous administration of atropine. He is of the opinion that clinical cases of pulmonary embolism give rise to reflex atelectasis more often than to infarction.

Swelling of Bronchial Mucous Membrane: Such a swelling may be the result of inflammation or of allergy. Allergic reaction as a cause of bronchial obstruction seems to be a possibility in some cases. Brown[11] found an edematous swelling causing obstruction of the right main bronchus in a patient whom he had bronchoscoped. Bergamini and Shepard[9] reported two fatal cases of bilateral total atelectasis, following anesthesia, in which the histological appearance of the alveoli and bronchioles was that of angioneurotic edema. Clarke[16] reported two cases of atelectasis complicating asthma in patients who had not been operated upon. Tucker[81] reported another case and expressed the opinion that the mechanism of obstruction was the same as in postoperative atelectasis. Clarke,[16] in reviewing the literature, found that more than 10 per cent of the reported cases of postoperative atelectasis had allergic histories and tests. In the light of these observations, it is very likely that a certain percentage of cases of postoperative atelectasis, in allergic patients, have an allergic factor, which, if not the actual inciting cause, must at least have its share in the production of the obstruction. The allergic reaction certainly furnishes the thick, tenacious mucus necessary for the formation of a mucus plug.

Mucus Plug: The concensus of opinion is that the usual cause of bronchial obstruction is a plug of thick, viscid and tenacious mucus. Such a plug may be formed from the normal bronchial secretions, from bronchial secretions which result from irritation or allergic reaction, or from material aspirated from the mouth and throat, for example, saliva, vomitus, pus draining from infected teeth and gums, pus draining from infected sinuses into the nasopharynx, or blood from upper respiratory operations. However, the mere presence of these fluid substances in the tracheo-bronchial tree cannot produce obstruction, for, as Coryllos[18] has said, "Bronchial obstruction cannot be produced unless two factors are present. The first is a more or less viscid bronchial secretion and the second, the inability of the lungs to expel it." Normally the tracheo-bronchial tree rids itself of accumulated secretions or aspirated fluid material by three means:[35]

1. The cough reflex.
2. The action of cilia.
3. Peristaltic movements in the air sacs, bronchioles and bronchi.

The tidal air passing through the tracheobronchial tree also aids by loosening and moving obstructions as it passes.[83] Any condition which interferes with this defensive mechanism will favor the retention and stagnation of secretions. That is exactly what happens in these cases. There is a markedly weakened respiratory force and a weakened or abolished cough reflex. Ciliary action is not abolished, except by excessively deep anesthesia or disturbed blood supply,[49] but its effectiveness becomes greatly reduced by the relatively large volume of accumulating secretions.

Most observers agree that the process is initiated by the pulmonary hypoventilation that results from the diminished respiratory effort. Muller, Overholt and Pendergrass[55] place primary significance upon this hypoventilation. They believe that the effects it produces are the determining factors that lead to obstruction of the bronchi and bronchioles. It acts in two ways to promote stagnation of secretions:

1. It reduces the size and motility of the bronchi.

2. It reduces the volume and force of the air that can be expelled from the lungs by coughing.[34, 55]

The result is the accumulation of normal secretions, inflammatory exudate or aspirated material. Yandell Henderson[37] pointed out that during shallow breathing many alveoli remain unventilated. The accumulating secretions may then obstruct the bronchioles which lead to these alveoli. Churchill[14] claims that even the normal bronchial moisture may cause obstruction if the breathing is shallow enough. Secretions of low viscosity may thus obstruct the finer bronchioles and produce scattered areas of lobular atelectasis,[12] but they could not obstruct the larger bronchi to produce massive atelectasis. Nevertheless, this mechanism may act indirectly to produce massive atelectasis by the spreading and confluence of many neighboring areas of lobular atelectasis. In order to occlude the larger bronchi, Band and Hall[6] by experiments on dogs, have shown that the secretions must have a viscosity corresponding to that of gum acacia of not less than 75 per cent concentration. Through the bronchoscope they saw the viscid material adhere to the bronchial wall. As the air passes through on inspiration and expiration there is a slight to-and-fro movement of the mass. During the dilatation of the lumen that accompanies inspiration the mass gradually moves down until it becomes arrested in a location where the lumen is of smaller calibre. Then partial, and finally, complete obstruction occurs. In clinical cases, according to Coryllos, the bronchial stasis and possibly also the anesthetic vapor or gas cause irritation of the bronchial mucosa. This leads to increased mucus secretion. Then secondary infection of these secretions by micro-organisms, including pneumococci, which descend from the upper respiratory tract, produces a bronchitis, accompanied by inflammatory exudate which becomes more and more viscid because of the production of fibrin by the pneumococcal infection. The resulting tenacious fibrinous exudate, plus inflammatory edema of the bronchi, produces obstruction of a lobar bronchus thus producing lobar atelectasis.

SECONDARY PREDISPOSING FACTORS

In the development of postoperative atelectasis, it is generally acknowledged that there are two chief factors, as I have pointed out:

1. Pulmonary hypoventilation, which acts as a predisposing cause.
2. Complete bronchial obstruction, which is the immediate inciting cause.

In addition, there are a great many secondary predisposing factors, some of them being so important that our etiologic picture would be far from complete without a clear understanding of the exact role played by each of them. They may be classified into two groups:

I. Those which act together to cause or augment the pulmonary hypoventilation.

II. Those which assist in the formation of bronchial plugs.

I. Predisposing Factors Associated with Hypoventilation.

1. Location and type of operation.
2. Elevation of diaphragm.
3. Reduced excursion of diaphragm.
4. Lowered vital capacity.
5. Increased intrapleural pressure.
6. Air beneath diaphragm.
7. Severity of operation.
8. Amount of postoperative pain, muscle spasm and muscle splinting.
9. Degree of obesity.
10. Intra-abdominal complications.
11. Abdominal binders.
12. Use of sedatives and hypnotics before and after operation.
13. Posture.
14. Old age.
15. Debilitating conditions.
16. Poor operative risks.
17. Sex.
18. Straining respiration.
19. Anesthesia.
20. Surgical technic.

II. Predisposing Factors Associated with the Formation of Bronchial Plugs.

1. Pre-existent pulmonary pathologic conditions, including allergy.
2. Hypersecretion of saliva and tracheobronchial mucus.
3. Preoperative use of atropine.
4. Aspiration of material from mouth, pharynx and nasopharynx.
5. Ineffective cough.

PREDISPOSING FACTORS ASSOCIATED WITH HYPOVENTILATION

One of the most important predisposing factors is the location and type of operation. Most of the cases of postoperative atelectasis occur after abdominal surgery, especially after operations in the upper abdomen.[27, 63] As a matter of fact, it rarely occurs as a complication of other operations.[27] Its incidence even varies in different upper abdominal operations. Sise[76] found it two and a half times as frequent after stomach as after gallbladder operations. During and after laparotomy the diaphragm is always greatly elevated and its excursion is reduced, and the expansion of the chest is diminished. Obviously, the result is a reduction in vital capacity. Powers[65] demonstrated, by actual measurements, that abdominal operations are followed by a marked reduction in vital capacity and that the greatest reduction and most prolonged period of recovery is after operations on the upper abdomen. Muller, Overholt and Pendergrass[55] also noticed this prolonged period of recovery after upper abdominal operations. Sise[76] observed that the operations which cause the greatest reduction in vital capacity are the ones which are followed by the greatest incidence of postoperative pneumonia. Thus it is clear that this reduced vital capacity, which means pulmonary hypoventilation, is an important reason why laparotomy makes the patient so susceptible to atelectasis.

VITAL CAPACITY AFTER OPERATION IN VARIOUS SITES

(The percentages refer to the per cent of preoperative level)

	Operation	Vital Capacity
Powers[65]	Extremities and rectum	No Reduction
Churchill and McNeil[15]	Abdominal	Reduced to 25-50%
Sise[76]	Lower abdominal	Reduced to 30-80%
	Upper abdominal	Reduced to 12-50%
Head[6, 76]	Lower abdominal	Reduced to 60-70%
	Upper abdominal	Reduced to 12-22%
	Lower thorax wall	Reduced to 66-86%
	Lower extremity	Reduced to 90%
Muller, Overholt and Pendergrass[55]	Upper abdominal	Reduced to 33%

An additional factor involved in the lowered vital capacity is an increase in intrapleural pressure. As a result of experiments on dogs, Prinzmetal, Brill and Leake[66] claim that ether anesthesia makes the normally negative intrapleural pressure less negative, therefore relatively increased, thereby producing less lung distention or relative lung compression. The intrapleural pressure is further increased by abdominal incision, pressure on the abdomen, and traction on abdominal viscera. They attribute this increase of intrapleural pressure to the elevation of the diaphragm.

Sise,[76] in comparing the x-rays of ten gallbladder cases before and after operation, found the right half of the diaphragm elevated and the adjacent lung tissue compressed. By fluoroscopy he noticed that the excursion of the diaphragm was reduced on both sides but especially on the right. Similarly Muller, Overholt and Pendergrass[55] observed this elevation of the diaphragm in the x-rays of 25 cases of upper abdominal surgery, the maximum range of motion being reduced to one-third of its preoperative range. The tidal excursion was reduced to one-half of its preoperative range and took place in the upper third of the possible range of motion, while before operation it had occurred in the lower third. They also found that roentgenograms made on the first postoperative day resembled those made preoperatively during full expiration. Band and Hall[6] believe that some diminution of diaphragmatic activity may be of reflex origin due to its relation to peritoneum, which exercises an influence over any movement that may be deleterious to itself or its contents. The first one to refer to weakened action of the diaphragm, in connection with atelectasis, was William Pasteur,[59] who, in 1890, reported the occurrence of atelectasis in cases of diphtheritic paralysis of the diaphragm. He believed this paralysis led to the development of atelectasis. However, Jackson,[40] in view of his own experience with diphtheria, believes that these cases were really due to bronchial obstruction by the diphtheritic exudate and not to paralysis of the diaphragm. By removal of these obstructions he prevents atelectasis in his own cases. This, however, is a different kind of case from the one under discussion. In diphtheria, with the rapid formation of firmly adherent and highly fibrinous exudate, no secondary factors are necessary to produce obstruction. It is comparable to the case in which an extrinsic foreign body has become lodged in a bronchus. On the other hand, in the cases under consideration, it is very likely that the weakened action of the diaphragm together with its high position, reducing the volume of the chest, and, thereby, the vital capacity and the force of respiration, sets the stage for the obstruction to develop.

AIR BENEATH THE DIAPHRAM

Overholt[55] reported that in 25 patients who had upper abdominal operations x-ray films of many of them showed air visible beneath the diaphragm. In experiments on dogs,[58] he verified the observations of others (Keppick, Melchoir and Melchio, and Wagoner and Krause) that the intraperitoneal pressure in the upper part of the abdomen is normally subatmospheric. He found that the intraperitoneal pressure decreases during inspiration and increases during expiration. Thus the subdiaphragmatic and intrapleural pressures fluctuate in the same direction during respiration. He suggests, from this observation, "that, normally, free diaphragmatic activity is favored by a subatmospheric pressure in the upper part of the abdomen. It would obviously be more difficult for the diaphragm to contract against a positive intraperitoneal pressure."

Using x-ray films, he observed the effect of: (1) injecting air into the peritoneal cavity and (2) laparotomy. In either case he observed, in every animal studied (1) an elevation of the diaphragm and (2) a diminution in diaphragmatic excursions. The reduction in the amplitude of the excursion amounted to approximately 50 per cent. "Under these conditions the diaphragm must move between a positive intraperitoneal and a subatmospheric or negative intrapleural pressure." Overholt does not claim that all of the diaphragmatic restriction after laparotomy is due to the presence of air in the peritoneal cavity, but he says that it may be partly responsible. This fact is fairly obvious.

SEVERITY OF THE OPERATION

Postoperatively, pain plays a very important part in restricting respiratory movements.[15, 39] The more severe the operation, the greater is the amount of postoperative pain, and the more pronounced are the results of the pain, which are:

1. Voluntary splinting of abdominal muscles in order to minimize pain.
2. Reflex muscle spasm which is even more effective in maintaining immobilization.
3. Voluntary guarding against coughing in order to minimize pain.

The splinting of the abdominal muscles and the muscle spasm markedly restrict the respiratory movements, especially those of deep and forcible expiration, such as cough. This further reduces vital capacity, and the lack of efficient coughing allows secretions to accumulate instead of being expelled. Sise[76] claims that these effects are more marked in the upper abdomen because the tension there is greater than in the lower abdomen, and the tension produced by inspiration is also greater because the costal margins are moving in opposite directions. The resulting greater tendency toward pain produces greater splinting and spasm of the muscles. Capelle (cited by Gius[31]) agrees with this muscle splinting theory and offers as proof the fact that he was able to reduce the spasm by the continuous injection of a local anesthetic into the wound and thus restore practically normal abdominal and thoracic respiration as well as vital capacity.

DEGREE OF OBESITY

Haight and Ransom[34] mentioned obesity as a factor in the causation of hypoventilation. This may act by making breathing more difficult due to the increased intra-abdominal content. If this is a correct view, then the greater the obesity, the greater would be the effect.

INTRA-ABDOMINAL COMPLICATIONS

Intra-abdominal complications, such as distention of the intestines,[34] acute dilatation of the stomach, and acute inflammatory complications, such as general peritonitis, lower the vital capacity. The first two act by encroaching upon the diaphragm. This was demonstrated in a case reported by Brunn and Brill.[12] In their patient, following an appendectomy, distention developed; the x-ray showed a high diaphragm with rotation of the heart; physical examination revealed diminished breathing, dullness and rales at both bases. Following an enema the x-ray was normal and the physical signs had disappeared. Peritonitis acts by increasing pain and distention.

ABDOMINAL BINDERS

Churchill and McNeil[15] showed that "the vital capacity of a normal individual was reduced 30 per cent by the mere application of a tight dressing over the right upper quadrant and costal border with the application of a binder such as is commonly employed to hold it in place." The more inelastic the dressing, the more it hinders respiration. Sise[76] points out that the effect of binders "is more marked in the upper abdomen than in the lower because more of the abdomen is usually covered by them and because in the upper abdomen they cover also the lower ribs."

USE OF SEDATIVES AND HYPNOTICS BEFORE AND AFTER OPERATION

Sedatives, administered before and after anesthesia, act by further reducing the pulmonary ventilation and by depressing the cough reflex. Both the barbiturates and the opiates depress respiration. In addition, the opiates depress ciliary action and the cough reflex, while at the same time increasing vagal tone with its resulting narrowing of the bronchi and increased viscosity of mucus.[10, 32] According to Brace,[10] these effects are especially dangerous in patients who have smokers' bronchitis or some other pulmonary inflammatory disease which depends on the cough reflex for the maintenance of an open airway. He says, "A state of inactivity of the cough reflex induced by drugs given 12 hours before the operation becomes a calamity when the re-establishment of the cough reflex is prevented before the evacuation of the accumulated secretion by additional drug therapy, administered one and one half hours before the operation."

POSTURE

The position of the patient has a definite effect on the ability to breathe. It has been shown that both the recumbent and the Trendelenburg positions embarrass the action of the diaphragm and reduce vital capacity.[6, 12, 75] During operations in which the patient lies on one side, the lower side is splinted by the weight of the body, thus restricting respiratory movements on that side. Secretions gravitate into the lower lung, thus providing the material for the formation of a plug.[12]

OLD AGE; DEBILITATING CONDITIONS; POOR OPERATIVE RISKS

Aged patients and patients with debilitating diseases are already suffering from pulmonary subventilation. They form a group which is highly susceptible to the action of additional factors that further decrease the respiratory effort. Waters[80] and his collaborators at the Wisconsin General Hospital observed that the percentage of postoperative pulmonary complications increased in proportion to the preoperative risk.

SEX

The sexual difference in the manner of breathing seems to predispose the male sex to atelectasis. It is common knowledge that normal respiration in the male is more abdominal than thoracic in type; consequently, after abdominal operations, the vital capacity is reduced more in men than in women.[34] As a result we should expect the incidence of atelectasis to be greater in men. Accordingly, King reported an incidence of pulmonary complications twice as great in men as in women.

STRAINING RESPIRATION

Straining respiration, from too light anesthesia or associated with coughing, grunting, moaning, etc., in which the vocal cords approximate and resist the passage of the expired air through the trachea, aids in the development of atelectasis. By experiments on dogs, Van Allen, Adams and Lindskog[82, 83] showed that straining respiration, in the presence of bronchial obstruction, helped to produce atelectasis, and the rate of atelectasis formation increased with increased straining. The increased pressure, on the air entrapped in alveoli whose airways are obstructed, leads to more rapid absorption.

ANESTHESIA

The anesthetic was for a long time, in fact, until approximately 20 to 25 years ago, regarded as the sole cause of all postoperative pulmonary complications, except pulmonary embolism. The irritating effect of ether, with its consequent stimulation of the secretion of large amounts of bronchial mucus, was well known; therefore, until more was known about other less obvious factors, it was quite natural to suspect the anesthetic agent very definitely. However, that the volatile anesthetic agents cannot be the sole cause is shown by two facts: (1) the occurrence of postoperative atelectasis almost exclusively as a complication of abdominal surgery, and (2) the fact that this complication occurs, after this type of surgery, just as often or oftener with spinal, local or regional block, in none of which direct irritation of the lungs occurs. In this connection, Yandell Henderson[36] calls attention to the fact that the pulmonary irritation caused by war gases leads to edema of the lungs instead of massive collapse. Taylor, Bennett and Waters[80] found the incidence of postoperative pulmonary complications at the Wisconsin General Hospital to be greater with spinal and regional block than with general inhalation anesthesia. Their results are as follows:

	Per cent
Cyclopropane	0.95
Ether, nitrous oxide and ethylene	1.36
Spinal and regional block	1.90

Mandl (cited by Elwyn[27]) reported the following incidence of postoperative pneumonia:

	Per cent
Ether	4.44
Local	4.39

Elwyn[28] reported the following incidence of postoperative pneumonia:

	Per cent
General	3.0
Local	2.7

Brace[10] reported an incidence of atelectasis with spinal equal to that with inhalation anesthesia. Brown (cited by Haight and

Ransom[34]) believes that atelectasis occurs more frequently after spinal than after inhalation or regional anesthesia because spinal inhibits the depth and force of the respiratory movements both during and for a time after the operation. Yandell Henderson believes that there is a diminution of body tonus with all types of anesthesia. With high spinal there may occur motor paralysis up to the fourth thoracic segment, thus producing paralysis of the lower intercostal muscles and resulting in hypoventilation.[72] This condition, acting concurrently with other predisposing factors, which are usually present with this type of anesthesia as well as with inhalation anesthesia, leads to the development of atelectasis.

The figures of Taylor, Bennett and Waters are most favorable to cyclopropane. Likewise, Burford[13] reported a low incidence of postoperative pulmonary complications after cyclopropane. His figures are as follows:

	Per cent
626 Abdominal operations	2.07
707 Non-abdominal operations	None

This percentage is greater than that of Waters and his collaborators but it is smaller than the average percentage at large, which is 4 per cent. Nevertheless, in spite of these favorable results, Burford found that, with cyclopropane, the incidence of such complications has increased more than should be expected when the anesthetic was of long duration (average two hours, one minute), especially for operations on the stomach. In all stomach cases the percentage rose to 21.7. A possible explanation of this occurrence may be found in the opinion of Brace[10] who strongly considers cyclopropane to be the most likely anesthetic agent to predispose to atelectasis "because of its marked parasympathomimetic action and depression of the respiratory center with decreased ventilation, inhibition of the cellular dehydrogenases, bronchial constriction, and increase in thick mucus." He considers ether to be less likely to lead to atelectasis, even though it produces an excess of secretions, because it does not produce bronchial spasm nor respiratory depression, the latter not occurring with ether until the stage of overdose is approached. This bronchial spasm in cyclopropane anesthesia is especially likely to develop when rushing a patient to a deeper level by suddenly increasing the concentration so that it becomes irritating.

When we consider what I have said, concerning inhalation anesthesia, we are almost inclined to let the pendulum swing to the extreme opposite direction, and, instead of condemning it unqualifiedly as was formerly done, to absolve it of nearly all blame. If we consider only the irritating effect of ether or cyclopropane on the bronchial mucosa, this policy might be somewhat plausible; but a careful analysis of other anesthetic factors involved forces us to revise our judgment so as to place, not only inhalation anesthesia, but every form of anesthesia in its proper category as an important, although, admittedly, not the only predisposing cause of postoperative atelectasis. These factors may be enumerated as:

1. The rapidity of absorption of anesthetic gases and vapors, as well as the physiological gases.
2. The general loss of muscle tonus.
3. The depth of anesthesia.
4. The duration of anesthesia.
5. The technic of the anesthetist.

THE RAPIDITY OF ABSORPTION OF GASES AND VAPORS

Coryllos[23] demonstrated in dogs that air in an obstructed area of lung takes six to 15 hours to be absorbed, while pure oxygen, carbon dioxide, or anesthetic gas or vapor are absorbed in a few minutes. Nitrogen and other inert gases take a long time to be absorbed (16 hours in the case of nitrogen) thus it is seen that the slow absorption of air is due to its high content of nitrogen. It is also clear that if an obstructed portion of lung contained a mixture composed of a high percentage of anesthetic vapor and oxygen, atelectasis could develop in a very short time following bronchial obstruction. This is what happened in the two cases reported by Bergamini and Shepard,[9] in which massive atelectasis developed soon after the operation. More recently Jones and Burford[42] reported four deaths from massive atelectasis of one or both lungs, proved by autopsy. These cases occurred rapidly after cyclopropane anesthesia. Jones and Burford explained that "with long continued shallow respirations, certain parts of the lungs, especially at the periphery, may not be ventilated at all. If, during the period of lack of ventilation, the gases in the resting alveoli consist of only absorbable oyxgen and cyclopropane, it is possible that they may be completely absorbed before further ventilation takes place. Patches of atelectasis may thus develop without an obstruction in any portion of the bronchial tree. This process, which may be termed creeping atelectasis, could go on, masked by the high oxygen content of the anesthesia atmosphere, until an entire lobe was involved. The first indication of it would appear when the mask was removed at the end of the operation. The patient would show all of the symptoms of respiratory embarrassment, such as marked cyanosis, restlessness and, in some instances, cessation of breathing." However, if the blood nitrogen of the supplemental and residual air are preserved, or if helium or hydrogen are added to the mixture, the absorption rate is lowered so that atelectasis takes longer to develop, thus allowing time for possible reaeration of the lung.

GENERAL LOSS OF MUSCLE TONUS; DEPTH OF ANESTHESIA

Yandell Henderson[36] has shown that there is a general loss of tone of all the muscles of the body during anesthesia. The resulting relaxation of the diaphragm may be so marked that x-ray pictures may show it to be 7 or 8 cm. higher than its normal position. Henderson claims, "This is as far or farther than the diaphragm moves under the deepest and most forcibly voluntary expiration" and this "results in such a diminution of the size of the thoracic cavity and such a deflation of the lungs that many minute deaerated areas are produced." He says that this condition is found after more than 80 per cent of all laparotomies when it is sought and that it is pure chance that massive atelectasis does not occur after all major operations, this chance depending upon mucus accumulating and blocking a major airway. He draws the conclusion that postoperative atelectasis, after any form of anesthesia, is due to diminished body tonus and its consequent deflation and collapse of the lungs. The deeper the anesthetic, the greater is the effect on respiration. This is probably the reason why operations which require deep anesthesia are followed by the greatest incidence of atelectasis. A. E. Guedel (cited by Burford[13]) expressed the opinion that 15 minutes of lower third plane anesthesia is more depressing than two hours of first plane anesthesia. In deep third plane anesthesia the intercostal muscles are paralyzed and are, therefore, sucked in during inspiration.[66] Of the thirteen cases of postoperative pulmonary complications reported by Burford[13] at least eleven of them had been brought to deep third stage anesthesia during the course of the operation. Similarly, high spinal has a greater incidence than low spinal,[36, 80] because, in high spinal, the lower intercostal muscles are paralyzed.

Burford is of the opinion that cyclopropane has an important advantage in that it can only rarely produce muscular relaxation beyond a certain degree; thus the degree of diminished muscle tonus is automatically limited.

Taylor, Bennett and Waters[80] reported the following results:

Anesthetic Plane	Percentage of Postoperative Respiratory Complications
General	
Analgesia	5.9
I	7.7
II	7.7
III	11.0
IV	15.0
Spinal	
Low (with intercostal activity)	6.1
High (with intercostal paralysis)	15.5

DURATION OF ANESTHESIA

The duration of anesthesia is a more important factor than is usually realized. The longer anesthesia lasts, the longer is the duration of the reduced muscle tonus with its deleterious effects, and the more opportunity there is for secretions to collect,[72] and for gases and vapors to be absorbed from alveoli whose bronchioles or bronchi are obstructed.

Taylor, Bennett and Waters[80] reported the following observations:

Duration of Anesthesia	Percentage of Postoperative Pulmonary Complications
Less than 30 minutes	2.9
From 31 to 60 "	7.7
From 61 to 90 "	13.2
From 91 to 120 "	15.2
From 121 to 180 "	19.3
From 181 to 240 "	30.3
Over 240 "	33.3
No data on time	4.6

TECHNIC OF THE ANESTHETIST

There is no doubt that the technic of the anesthetist is of especial significance since many factors, under his control or supervision, are involved. A badly administered anesthetic, with straining respiration, obstructed airways, coughing, and retching, will favor the development of atelectasis. Reduction of the carbon dioxide content of the anesthetic mixture to too low a percentage, by means of the carbon dioxide absorber, will produce shallow breathing. Bronchial irritation, from excessive concentration of ether vapor or cyclopropane, will play a minor role, but, nevertheless, its effect will be added to that of the other factors. This irritation may produce a bronchitis,[27] which is a link in the chain of events that leads to atelectasis. This irritation can be kept at a minimum by the well trained anesthetist. Keeping a patient too long at excessively deep levels, instead of permitting him to return to lighter levels compatible with the surgical requirements, will prolong unnecessarily a high degree of diminished muscle tonus. However, in spite of all that has been done, these complications continue to occur even where skilled anesthetists are employed.[71]

SURGICAL TECHNIC

No less important than the anesthetic technic is the surgical technic. Carelessness on the part of the surgeon or his assistants can do much harm. Large, traumatized wounds, infected wounds, excessive traction on abdominal viscera, rough retraction on abdominal muscles, especially upward retraction that makes pressure on the liver and diaphragm, intra-abdominal packs which interfere to some extent with the descent of the diaphragm, leaning and resting on the patient's abdomen and chest,[66] and dressings that restrict respiratory movements all take part in the production or maintenance of hypoventilation.

PREDISPOSING FACTORS ASSOCIATED WITH THE FORMATION OF BRONCHIAL PLUGS

Pre-Existent Pulmonary Pathologic Conditions: The preoperative presence of inflammatory diseases is not necessary for the development of atelectasis. Scott[73] reported their absence in most of his cases that had been checked by physical examination and x-ray before operation. Nevertheless, the presence of a pulmonary infection will favor the production of excessive thick secretions.

The manner in which allergy favors atelectasis has already been described in the discussion of obstruction of the bronchial tree by swelling of the mucous membrane.

Hypersecretion of Saliva and Tracheo-Bronchial Mucus: Ether and cyclopropane cause a hypersecretion of saliva and tracheo-bronchial mucus. The former may find its way into the tracheo-bronchial tree by aspiration.

Preoperative Use of Atropine: Atropine has been accused of concentrating the secretions so that they become thick, viscid and tenacious[50, 56] and are then difficult to expel. On the other hand, it has been credited with beneficial effects. Brace[10] claims that it abolishes many of the adverse effects of morphine, especially the increased bronchial muscular tone, the secretory action and the depression of the respiratory center. Likewise, de Takats[25] claims that it inhibits autonomic reflexes and thereby prevents the occurrence of bronchospasm.

Aspiration of Material from Mouth, Pharynx and Nasopharynx: Aspiration is probably an important source of obstructing material. Schlueter and Weidlein[71] believe that this occurs very often under general anesthesia. Lemon,[49] in experiments on dogs, observed that aspiration of material in the mouth occurred, whether anesthesia was light or deep, when the animals were inclined with head up and even in the horizontal position. As the inclination proceeded, with the head end moving downward, less and less aspiration occurred, until the marked Trendelenburg position was reached, when none occurred. Other experiments showed that aspiration occurs also when large amounts of fluid are suddenly vomited. In the same way, aspiration of infected material, such as pus draining from infected sinuses into the nasopharynx, may occur. Similarly, blood from operations in the nose or pharnyx, may be aspirated.

When a patient lies on one side, there is a tendency for secretions to collect on the dependent side.[12] They may pass from the upper to the lower lung or even from one lobe to another of the same lung.

Ineffective Cough: Cough may act in one of two ways: (1) it may clear the tracheobronchial tree by bringing about the expulsion of secretions or other material, (2) it may drive them deeper so as to favor obstruction. During the act of coughing, the inspiratory rush of air may carry material deeper; and the expiratory phase may force some of it deeper, or spread it to other areas of the lung.[5] It is only material in the larynx, trachea, or larger bronchi which can be expelled by coughing; and then, only if the force of expulsion exceeds that of adhesion. Material below the level of the larger bronchi tends to be forced deeper into the lung.[82, 83]

CORRELATION OF ETIOLOGIC FACTORS

All available evidence demonstrates the fact that postoperative atelectasis is the result of a sort of chain reaction, involving a relatively large number of etiologic factors, the most important of which are pulmonary hypoventilation and bronchial obstruction. Both of these are present in every case, with the possible exception of a few cases in which the mechanism may be that described by Jones and Burford, and in which atelectasis in the peripheral alveoli follows hypoventilation without actual obstruction. In the vast majority of cases, however, there is obstruction by a mucus plug, the removal of which relieves the condition. Thus it is evident that bronchial obstruction is the actual inciting cause and hypoventilation is the factor which sets the stage for the obstruction to develop. In turn, these two factors are produced by the collective action of a number of the other factors, acting in unison or consecutively. All of these secondary factors will not be found in any one case, but enough of them are always present to induce the undesired result.

COURSE OF EVENTS AFTER DEVELOPMENT OF ATELECTASIS

Once atelectasis is established the further course depends upon the elimination or continuation of the obstruction. If the obstructing mucus is expelled by coughing, or by bronchoscopy the lung will become reaerated and the condition will thereby be terminated. If coughing only partially removes the obstruction the lung may become partially aerated. It is also possible for coughing to clear some bronchioles or bronchi and, at the same time, to spread mucus to others, resulting in newly obstructed areas, thereby reaerating one portion of lung and causing atelectasis in another portion. In like manner, a previously reaerated area may again become atelectatic. Thus the physical signs may change according to the change in location of the obstruction.[18]

If the obstruction is maintained the result will be comparable to that which occurs when the duct of a glandular organ is obstructed,[23] and the course will depend upon the presence or absence of micro-organisms in the obstructed bronchus.[17] If there are no microbes, the case will remain as simple atelectasis. If infection is present, the micro-organisms will invade the collapsed alveoli, and the further course will be determined by the kind of micro-organisms present and their virulence. Many patients have a non-virulent type IV pneumococcus in their respiratory tracts. If this is the only organism present, simple atelectasis will still result, as explained by Coryllos. If the pneumococcus is more virulent the exudate will be richer in fibrin, and therefore more viscid and more difficult to dislodge. The result will be the development of a pneumococcic cellulitis in the alveoli, and the degree of toxicity of the disease will be determined by the number and virulence of the organisms present.[19] If the virulence is low, a mild pneumonia will be the result; and if it is high, a severe pneumonia will result. The variety of pneumonia that develops depends upon the type of atelectasis present. Lobular or patchy atelectasis leads to bronchopneumonia and lobar atelectasis leads to lobar pneumonia.[18]

If, instead of the pneumococcus, the infection is by other bacteria, such as *B. influenzae,* staphylococcus or streptococcus, which produce less viscid exudate, the resulting condition is more likely to be lobular atelectasis followed by lobular pneumonia.[22] If the infection is by a highly virulent pneumococcus the resulting condition is more likely to be lobar atelectasis followed by lobar pneumonia.

Coryllos[21] is convinced that the mere presence of infection in the tracheo-bronchial tree is not sufficient for the production of pneumonia; but that it may result in bronchitis, the exudate of which must occlude a bronchus in order for pneumonia to develop. Thus pneumonia develops as a complication of atelectasis; and postoperative atelectasis and postoperative pneumonia are two phases of the same condition. Elwyn[27] and Band and Hall[6] also are in accord with this conception of postoperative pneumonia.

Furthermore, the experimental and clinical observations of Allen,[3] Joannides,[41] Smith,[77] Brunn and Brill[12] and Jackson[38] indicate that if the atelectatic alveoli contian anaerobic organisms (spirochetes, fusiform bacilli, streptococci, and vibrios) lung abscess may develop as a complication.

SUMMARY

1. Atelectasis is the most common postoperative lung complication.

2. It is most frequent after abdominal operations, especially those in the upper abdomen; and rare after other operations.

3. It is very often diagnosed as postoperative pneumonia.

4. It is the precursor of postoperative pneumonia and some cases of postoperative pulmonary abscess.

5. It is initiated by a great many interacting factors which lead to the production of pulmonary hypoventilation and bronchial obstruction. The hypoventilation sets the stage for the formation of a mucus plug.

6. Hypoventilation is increased by postoperative pain, muscle spasm, muscle splinting, obesity, tight abdominal dressings and bindings, intra-abdominal complications, the use of sedatives and hypnotics before and after operation, old age, debilitating conditions, the horizontal and Trendelenburg positions, and deep and prolonged anesthesia.

7. The formation of an occluding plug is favored by the preoperative presence of inflammatory or allergic pulmonary disease, hypersecretion of saliva and tracheo-bronchial mucus, thickening of bronchial secretions by the preoperative use of atropine, aspiration of material from the mouth and throat, and ineffective cough.

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MALARIA

AN EPIDEMIOLOGIC STUDY[*]

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Major, M.C., A.U.S.

Concern has been expressed by both medical and lay groups that the return of troops fron endemic malarious areas would react unfavorably upon the health of continental America. This point of view is mainly based upon the assumption that returning service men may introduce the disease into areas of this country in which malaria has been absent for many years; and too, augment its incidence and severity in other areas where it is normally endemic.

To examine this contention, it seemed feasible to undertake an epidemiologic survey in an area where malaria vectors exist, and where a rigid military control program is in operation. Then, under controlled environmental conditions of camp life, factors which might augment the malarial rate for this community, could be ascertained.

The material for this study was obtained from the medical admissions to the Station Hospital Annex, at the Prisoner of War Camp, Camp Livingston, Louisiana. The period surveyed was from 7 July 1943, the date of arrival of the prisoners of war, to 7 July 1944. The personnel of this camp included German prisoners of war, captured in the North African and Sicilian theatre of operations, and enlisted men of the guard detachment from various parts of continental United States. The prisoners of war had all received malarial fever suppressive therapy (0.1 gm. of atabrine per day) during their tenure of service in their respective areas. No suppressive treatment was given during the period of study.

STATISTICAL DATA

A. Number of patients having proven malaria: 148 (3.9 per cent of the mean prisoner of war strength.)

B. Number of attacks, with period of hospitalization: 272. N. B. 46 patients having a total of 50 attacks of so-called "fever of undetermined origin" (clinical malaria but not demonstrated by blood smear) in the period under study were not included in this analysis.

C. Percentage of malarial fever cases from total admissions to the medical service: 37.7.

Percentage of malarial fever cases from

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[*] The source material for this paper was gathered during the author's tenure of service at the Station Hospital Annex, Camp Livingston, Louisiana.

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total admissions to the hospital (medical and surgical): 25.5.

D. Origin of infection in those giving a prior history of malarial fever:

North Africa: 30
Sicily: 4
Russia (Crimea): 2

Total number of attacks outside continental United States: 115.

E. History of two or more attacks: 64

1. Maximum number of attacks in one soldier: 6

2. Average interval between attacks: 56 days

(a) longest interval period: 205 days
(b) shortest interval period: 18 days

F. Distribution of attacks by months.

July, 10; August, 10; Sept., 27; Oct., 17; Nov., 10; Dec., 9; Jan., 18; Feb., 30; March, 45; April, 48; May, 34; June, 14.

G. Most recent primary attack of malarial fever: 5 July 1944.

H. Type of Plasmodium demonstrated: P. vivax. N. B. In three patients P. falciparum was demonstrated: however, in subsequent attacks P. vivax was found to be the causative agent.

I. Average days of fever during period of hospitalization: 2 days.

J. Patients showing liver enlargements: 3; patients showing spleen enlargements: 19.

K. Malarial fever admissions to the Station Hospital, Camp Livingston, Louisiana. "Station Hospital serves only American Personnel in Camp Community."

(1) Total admissions in the period under study: 23,221.

(2) Total number of acute malaria (primary attacks): 11.

(a) origin of infection, Louisiana area: 8.

(b) origin of infection outside the continental United States: 3.

(3) Total number of chronic malaria (relapses): 51.

(a) origin of infection: Louisiana area: 6.

(b) origin of infection outside the continental United States: 45.

DISCUSSION

The possible introduction of more virulent strains of malarial parasites from large numbers of service men, coming from hyperendemic areas might offer a serious threat to public health in this country. A perusal of our data for the year studied, shows that despite the presence of vectors for the transmission of the disease (K) there were no sporadic outbreaks of malarial fever, either amongst the prisoners of war, guard detachment, or the surrounding camp community. This is substantiated by our data (A & D) relative to the occurrence of malaria among the prisoners of war (3.9 per cent) who served in moderate to highly endemic malarious areas, and the admissions for malaria fever at the Station Hospital which serves the American personnel (K). Indeed, not one enlisted man affilated with the guard detachment developed malarial fever in the period of study. The author is of the opinion, that the rigid supervision over malarial control practiced at the camp and the surrounding areas, prompt diagnosis and treatment, contributed to keep the malaria rate under control.

Of the 148 prisoners of war studied, 36 gave a history of having had malarial fever outside the limits of the continental United States. (D). When questioned, these prisoners could not state with certainty whether the diagnosis was corroborated by blood smears. However, they manifested clinical improvement under atabrine medication. Though the incubation period of malaria may vary with the type of plasmodium, the resistance of the individual, it is stated to be as early as 10 days, and may extend to seven months (Craig and Faust, Clinical Parasitology, pages 205-206). In our series (G), the most recent primary attack of malarial fever occurred 5 July, 1944. It cannot be stated with certainty that even this attack (prisoner of war) had its origin in this area, for delayed primary attacks may occur as long as a year after cessation of suppressive medication.

Following diagnosis, the patients were put uniformly under routine malaria management as suggested by S.G.O., Circular

Letter 153, 5b, dated 18 August, 1943. None of the patients under study showed untoward effects to atabrine therapy. In general, our experience with atabrine medication (B,C,D,E,F,I,J) confirms the fundamental disadvantages inherent in the available drug therapy (atabrine), namely (1) drugs cannot be relied upon to prevent the development of malaria following the bite of the infected mosquito; (2) they do not eradicate the disease. The taking of drugs serves to check the multiplication of the parasites sufficiently, so that most protected persons will not develop active malaria during the period which they are ingested.

SUMMARY AND CONCLUSIONS

An epidemiologic study was undertaken of 272 attacks of malarial fever in 148 prisoners of war (mean prisoner of war camp strength ±5000) for a period of one year. The analysis revealed that there were no sporadic outbreaks of malarial fever either amongst the prisoners of war, guard detachment, or camp community. In fact, not one enlisted man of the guard detachment contracted malaria in the period under survey. One primary attack of malarial fever in a prisoner of war, occurring 5 July, 1944, may have had its origin in this area, although this too may be a point of contention. The low incidence of malaria in this area is attributable to the malaria control program practiced at this military installation, rapid diagnosis and treatment.

Where rigid control is exercised, the disease is kept local and transient.

Though the utopian achievement of control and eradication of the disease has not taken place, one may be reasonably optimistic that the new knowledge gained in this war in technic of mosquito control and treatment will tend to clear up much of the disease already existing on this continent.

Grateful acknowledgement is made to Major Henry C. Brown, M.A.C., Registrar, Station Hospital, Camp Livingston, Louisiana, and to the Louisiana State Dept. of Health in the preparation of this article.

SPINAL EPIDURAL ABSCESS

REPORT OF A CASE*

J. A. COLCLOUGH, M.D.†
NEW ORLEANS

Acute spinal epidural abscess constitutes a grave surgical emergency. Unrecognized and without early surgical intervention it produces compression, interruption of function, and irreversible damage to the spinal cord. The results of such a transverse myelitis are paraplegia, retention or incontinence, decubitus, infection and death. Those of us who have not seen the development of these consequences should keep the possibility of spinal epidural abscess in mind when examining a patient with a possible meningitis, acute poliomyelitis or encephalitis.

According to Boyce,[1] all available cases of spinal epidural abscess were analyzed by Dandy[2] in 1926. Dandy reported 27 cases, including one of his own. Four of these cases were operated upon and two survived. All other cases were fatal. In 1933 Bunch and Madden[3] studied 19 additional cases and reported two of their own. Three of these were not operated upon and died. Of 16 operated upon, four died, four were left with paraplegia and eight made complete recovery. Echols[4] has reported four cases with recovery in three of the four. To the best of my knowledge, the latest written upon the subject is an article by Grant[5] in June of 1945, in which he reports 14 cases and states that over 200 cases are reported in the literature. I do not believe that this comparatively rapid increase in number represents an increase in incidence, but, rather, that it means increasingly early diagnosis and treatment. In spite of recognition as early as 1820,[6] and since there is only a modest number of such cases available in the literature, a great number of deaths must have resulted from unrecognized acute spinal epidural abscess since that date. The object of this report is therefore twofold: (1) to increase our consciousness in regard to this clinical entity, and (2) to add one more case to the literature.

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*Read before the Scientific Meeting of the Orleans Parish Medical Society, June 10, 1946.

†From the Surgical Service of Tulane University School of Medicine and the Charity Hospital of Louisiana in New Orleans.

CASE REPORT

N. G., aged 16, a white male, was admitted to the Charity Hospital of Louisiana in New Orleans on November 11, 1944 with the provisional diagnosis of poliomyelitis. His chief complaint was pain in the back and in the right side.

The family history and past history were irrelevant. The review of systems was negative except for the symptomatology of his present illness.

Present Illness: Fifteen days prior to admission he had developed fever and pain in his back and right side. There were no other symptoms. Neurologic examination was said to have been negative two days previous to the date of admission, but upon admission he had developed muscle spasm and paralysis of the right lower extremity. He had been vomiting and was incontinent.

Physical Examination: The temperature was 100.2° F. The patient did not appear to be acutely ill, but lay flat in bed without moving. He was rational and cooperative. The head was negative. He was unable to flex his neck because of the pain it caused in his back. The neck muscles themselves did not seem spastic. The heart and lungs were negative. Slight rigidity was present in the abdominal muscles, and the bladder was greatly distended. There was slight urinary incontinence. The extremities were negative except for the right lower extremity which was flaccid and externally rotated. There was complete paralysis of the right thigh and leg except for a slight degree of abduction, adduction, internal and external rotation. The abdominal reflexes were absent. The tendon reflexes of the upper extremities were normal. The left patellar reflex was hyperactive, and the right patellar reflex was hyperactive. Both ankle jerks were hyperactive, with slight clonus on the right. No positive Babinskis were found. Kernig's sign was present bilaterally. The blood count was 30,500 white blood cells with 93 per cent polymorphonuclear leukocytes. The impression of the examining physician was acute poliomyelitis.

On November 13, 1944, two days after admission, a spinal tap was done, the level not being recorded. The fluid dripped very slowly and about 1 c.c. of xanthochromic fluid was aspirated which clotted immediately when put in a test tube. The protein content was reported to be 1080 mg. A request for consultation to neurology was answered by Dr. Walker Thompson with the following findings: "Paralysis of the right leg, spasticity of the back, hamstring and other muscles bilaterally, weakness of the right arm, hand, absent abdominal reflexes, incontinence of urine and feces. The cell count done in Lafayette, Louisiana Charity Hospital was 94. I do not believe the patient has poliomyelitis. There is evidence of pressure caused by a lesion apparently located somewhere between the fourth and seventh thoracic segments. Impression, possible epidural and sub-dural abscess. Patient should be seen by neurosurgery. Decompression may be necessary."

The patient had previously been seen by physicians on the orthopedics service, who regarded the diagnosis of poliomyelitis as questionable. Packs over the neck, chest, abdomen, upper and lower extremities were advised. It was also suggested that the patient be watched for signs of bulbar involvement.

Shortly thereafter the patient was seen by me and the following findings were recorded: There was a history of pain in the right side beginning ten days ago accompanied by fever. There was weakness of the legs with rapidly advancing symptoms of cord involvement for the past four days; namely, weakness of the lower extremities, bladder and rectal disturbance (retention and incontinence), weakness of both upper extremities. The biceps reflexes were present and equal; the triceps were absent. The knee jerks were absent and the ankle jerks were present and equal. I was unable to elicit any plantar reflex. There was rather marked stiffness of the neck. Exertion of pressure over the spinous processes from approximately the fifth thoracic to the ninth thoracic produced pain locally which was referred to the chest anteriorly. Deep sensation was partially absent in the lower extremities. There was no clear-cut sensory level, but an area of hypesthesia was noted in dermatomes T 6 to T 10. Attempted spinal puncture at the third lumbar interspace was unsuccessful, and at the second lumbar interspace aspiration resulted in withdrawal of a small amount of blood-tinged, purulent material. A diagnosis of spinal epidural abscess was made and the patient was transferred to the neurosurgical service immediately.

Operation: On November 13, 1944, under general anesthesia, a dorsal midline incision was made from the spinous process of the fourth to that of the eighth thoracic vertebra. The soft tissues were bilaterally separated from the spinous processes and laminae by means of periosteal elevator and sharp dissection. The wound was held open by means of self-retaining retractors. The spinous process and laminae of the sixth thoracic vertebra were removed, and as the ligamentum flavum was being removed, creamy-white pus which was thick in consistency was encountered. There was no odor. The spinous processes and laminae of the fourth and fifth, seventh, eighth and ninth thoracic vertebrae were then removed and the abscess was uncovered throughout this extent. There was no granulation tissue noted. Pus filled the entire area. The limits of the abscess had not yet been reached. By means of a catheter passed epidurally, an attempt was made to determine the extent of the abscess, but judging from the fact that pus was obtained in this manner for a distance of about three inches beyond the unroofed area both approximally and distally, it was deemed inadvisable to attempt to uncover the entire extent of the abscess. Epidural rubber tissue drains were placed above and below the exposed area, and throughout the extent of the uncovered dura. Following this, the soft tissues were approximated loosely using cotton as interrupted sutures. The operation was two hours and two minutes in duration.

Penicillin therapy was instituted postoperatively and was given through Nov. 23, 1944. Fever persisted until Nov. 17, (four days postoperatively) when the temperature was almost normal throughout the day. Improvement in motor function was slow. Twenty-six days after operation (on December 9, 1944), he began to feel the desire to defecate and urinate, and could accomplish these acts voluntarily. On December 11, 28 days after the operation, he was able to flex and extend his left leg and foot, and to adduct and abduct the left thigh. Six weeks after operation he was able to flex and extend the right leg and thigh, and could evert and invert the right foot. On the following day, (December 29) he could stand, but was unable to walk without the aid of crutches. He was discharged on this date to the physiotherapy clinic. Cultures of the material obtained by aspiration produced staphylococcus aureus pyogenes. Culture from the abscess showed the same organism. The blood culture was negative. The serology was also negative.

On October 22, 1945, the gait was spastic with the greater amount of spasticity being present in the right lower extremity. There was no appreciable muscular atrophy. The patellar reflexes were exaggerated, the right being greater than the left. The ankle jerks were hyperactive, the right being more so than the left. There was some muscular weakness of the right lower extremity, but there was a remarkable degree of recovery of muscular strength on both sides. The Babinski was bilaterally positive. There was no impairment of pain sensation. Proprioception was unimpaired. The deep muscle sense was somewhat impaired on the left. Touch was unimpaired. The operative wound was healed, but there was one small sinus in its lower third where a buried suture had been removed that morning. Vibratory sense was unimpaired. Sustained clonus was present on the right. The abdominal reflexes were absent.

On April 21, 1946, eighteen months after operation, he was again examined. At that time the gait approximated the normal. He stated that he could walk much better, and was now able to dance. Other than some weakness of the right leg, he had no complaint. Neurologically some spasticity was still present. There was some muscular weakness in the right lower extremity. The knee and ankle jerks were hyperactive. Ankle clonus was present on the right. There was no neurological or functional impairment of the upper extremities.

It is now eighteen months since operation, and I believe recovery has about reached the maximum.

DISCUSSION

There is nothing remarkable about this case except the size of the abscess. From passing the catheter superiorly at the time of operation, I know that it extended superiorly to the level of the first thoracic vertebra. Neurologically the cord was affected to the fifth cervical segment or the level of the fourth cervical vertebra. Because pus was obtained by aspiration at the second lumbar interspace I know that it extended inferiorly to the upper border of the third lumbar vertebra. In other words, the lower part of the cervical, the entire thoracic, and the greater portion of the lumbar epidural space was involved. It is also noteworthy that this patient was operated upon more than 48 hours after he became almost completely paraplegic, yet the degree of recovery is almost 100 per cent.

Lumbar puncture in cases of suspected epidural abscess must be done with the greatest of care to avoid the possibility of a needle which has traversed an abscess penetrating to the subarachnoid space. As soon as the interspinous space is reached aspiration should be attempted. If no pus is obtained, the needle should be inserted successively deeper, a millimeter at a time, and aspiration should be attempted as each new depth is attained. This should be repeated until pus or cerebrospinal fluid is obtained by aspiration. If the former is found, the diagnosis is established; if the latter, the Queckenstedt quickly reveals it.

The diagnosis of spinal epidural abscess is usually not too difficult if one thinks of it as a possibility. There is usually a history of a pyogenic infection somewhere in the body. However we were unable to obtain one in this case. Pain in the back at any level, tenderness over the spine, root pain, fever, elevation of the leukocyte count, weakness of any extremity with the development of upper motor neuron signs form a picture which is quite clear. The onset of weakness of the extremities and bladder disturbance makes spinal puncture mandatory. The laboratory findings of increased protein and cell count are confirmatory evidence. The existence of block makes immediate operation an urgent necessity.

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DISCUSSION

Dr. Dean H. Echols (New Orleans): Surely this is the most "epidural abscess conscious" county medical society in America. About six or seven years ago Dr. J. O. Weilbaecher called our attention to this entity. A year or two later I brought two postoperative cases of epidural abscess to this platform. Since then one of us has published the accumulated cases from Charity Hospital in one of the surgical journals. I am glad Dr. Colclough took up the torch because epidural abscess is too important a disease to go unrecognized. If the diagnosis is made because physicians are thinking about that possibility the patients can be cured. I thought for a time that the advent of chemotherapy would put an end to cases of epidural abscess but they have continued to turn up from time to time just the same. One of the important points that Dr. Colclough brought out is that we can not rely on chemotherapy alone to cure all infections; prompt and adequate surgery is also frequently needed.

Dr. Walker Thompson (New Orleans): This condition is particularly important because it is a medical or surgical emergency that can be taken care of and can be diagnosed without very much difficulty. Of course sometimes it looks like a cord tumor syndrome, an extramedullary cord tumor, starting out with involvement of the roots and pain at the level of the abscess. Rather rapidly it causes pressure upon the cord and paraplegia. The thing that gives it away particularly is the tenderness over the vertebra which I believe is more marked than in cord tumors. There may or may not be severe meningeal signs although the patient is not going to be prostrated like we see in meningitis. I do not believe I have anything else to add except that we should teach this syndrome since it can be taken care of with good results as we see in this case.

Dr. Colclough (in closing): I wish to thank Dr. Echols and Dr. Thompson, both of whom have been my mentors, for their discussion and kind remarks.

The first case of epidural abscess I saw was shown to me by Dr. Echols and chemotherapy cured that particular patient. We have also seen an occasional case that clinically we were sure was brain abscess, cured by the same means. By the immediate institution of drug therapy, either one of the sulfonamides or penicillin, there will possibly be a decrease of incidence of spinal epidural abscesses which come to operation. We must be conscious of this condition. Bear it in mind and if it does not yield quickly to chemotherapy, surgical intervention must be instituted.

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THE METRIC SYSTEM

The American Medical Association has adopted the metric system entirely, not only for all of its publications but also for its New and Nonofficial Remedies. The Committee on Revision of the Pharmacopeia will do likewise, employing only the metric system. The Pharmacopeia Committee rightly points out that metric doses in the past have been based upon the apothecary dose and its equivalent. As an example, the usual dose equivalent for a half grain was considered to be 32 milligrams and for five grains 325 milligrams. It is the point of good sense however, to use round figures in the preparation of metric system dosage so that instead of 32 milligrams drug manufacturers will use 30 milligrams and instead of 325 milligrams will use 300 milligrams for the actual dosage. The same relationship will be followed by the pharmacopeia.

Many of the older doctors and even young physicians seem to have a fear of the metric system. Perhaps if round numbers are used this fear may disappear. Actually the present day physician uses the metric system almost interchangeably with the apothecary's. The physician will order 500 c.c. of a solution to be given intravenously and then he will put the amount of the drug he wishes to be given in this solution in the apothecary system. In prescribing most of the common drugs the apothecary system is employed, yet when it comes to the using of arsenicals the metric system is the one that is almost universally employed. Of neoarsphenamine a gram more or less will be ordered for the patient and the apothecary system will be completely forgotten.

The metric system of course has so many advantages that they need not be recounted. It is the universal dosage system employed throughout the world. From the action of the A. M. A. it behooves the physician to learn this particular system. Elsewhere in the Journal is published a table of metric dosage with approximate apothecary equivalents prepared by the Committee on Revision of the Pharmacopeia. This may be used for ready reference in prescribing in the future.

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DIPHTHERIA MORTALITY

For many years it was the custom of the Journal of the American Medical Association to review the diphtheria deaths in the large cities yearly. However, on account of the exigencies of the war and because diphtheria, in incidence, was diminishing markedly, no figures have been published since

1942 until they appeared in the July 27 number of the Journal this year.

First, a word as to the one city of Louisiana, New Orleans, which is reported upon. There has been an increase in the city in diphtheria mortality from 2.5 per hundred thousand population to 3.4. Only Fort Worth, in the cities in the west-south central states, exceeds New Orleans in its diphtheria mortality.

It should be noted that there are many cities in which there have been no deaths from diphtheria whatsoever. There were 28 such cities.

It should be pointed out that in some of the cities which had a fairly large death rate many of the children who died were imported into the city and were not residents thereof. It furthermore should be observed that there has been an increase in the actual number of deaths throughout the cities of the country. There is a tendency for this disease at the present time to be relatively more prevalent among the higher age groups. This would bespeak the influence of immunization of the small baby and a failure to give booster treatments to the older children at the time that they are admitted to school and from time to time thereafter until they obtain maturity. Doctors have been taught well the importance of immunizing the infant but they should not forget that booster protective treatments should be given from time to time, not only with this particular disease, diphtheria, but also with other diseases in which passive immunity is induced.

THE NEED FOR HOSPITAL BEDS IN LOUISIANA

Comment has been made in a previous editorial on the large amount of money that is spent by the State of Louisiana for the maintenance of general hospitals. It was pointed out that of most states in the Union, Louisiana pays for medical and dental care in a greater amount per capita than any other state except for several of the small New England states, New York and Colorado. A little over 67 per cent of the total expenditures are spent for the general medical care of sick Louisianians which is almost 25 per cent more than any other state pays from its total expenditure for medical care. It was noted also that some 48 per cent of all hospital income is provided from taxes appropriated by our State Legislature as contrasted with the next state, California, of which only 37.8 per cent of hospital income is derived from taxes. From these figures compiled from "Medical Services by Government" published by the Commonwealth Fund a few months ago, it is obvious that Louisiana has been more than generous in taking care of its sick citizens.

The figures that have been given bring up the question as to the need and necessity of further extension of state supported hospitals in Louisiana. The United States Government is prepared to give untold millions to provide hospitals and hospital care for the citizens of the country as a whole but it must be borne in mind that some states have spent and are spending but little; Alabama for example expends only 47 cents per capita for medical care, while Louisiana spends $1.88. Other sections of the country may need additional hospital facilities but merely because the United States Federal Government offers the opportunity of getting a few million dollars which has to be met with an equal contribution by the state should not make the state authorities grab at this money. The state must put up a very substantial amount but the real cost of hospitalization is the continuous running of the institution, its maintenance and its upkeep. The initial outlay is minimal as contrasted with the ultimate outgo of monies for the maintenance of an institution. Certainly it does not seem right, fair nor just that our taxpayers should be assessed additional taxes in order to provide general hospital care which already is more generous than is given by practically any state in the country.

The addition of tuberculosis facilities may be necessary but again we would question the need for any great outgo of money for such facilities. The Pines Sanitarium in

Caddo Parish, for example, is only 35 per cent occupied. The death rate from tuberculosis in Louisiana has gone down by leaps and bounds from a total of 84.5 in 1930 to 45.3 per 100,000 in 1945. The negro rate has diminished almost 50 per cent as has that of the white rate. Tuberculosis is being conquered and great expenditures would seem unnecessary for the care of patients with this disease.

The state is behind most states in the provisions made for psychiatric services but psychiatric cases are diminishing now that syphilis is being so adequately controlled and other therapeutic measures are proving efficacious in prevention and in the care of the psychiatric patient.

It would seem that the burden of proof is on those who advocate a constitutional amendment to increase our state debt by many million dollars. Already Louisiana's expenditure for general hospital care is extremely liberal; tuberculosis is a disease which is decreasing in incidence and in which furthermore since the introduction of pneumothorax treatment of the disease more and more patients are being treated as ambulatory cases. Merely because the Government offers monies is no reason to seize upon this money with the idea that it will be cheap. In the long run the initial Federal grant will be small compared to the total money that will have to be spent in the future by the state to maintain the contemplated institutions.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 DUES

During November notice will be sent to members concerning dues for 1947 which should be paid in December of this year. Amount of dues will be continued at $25.00 and this with dues for membership in the parish or district society should be sent to the secretary of the component organization.

Total active members in the State Society for 1946 is 1576, of which 251 were entitled to deletion of dues because of military service. These figures show an increase of 152 paying members over number for last year. Dues were deleted for 339 members in 1945 whereas this number was decreased by 88 members this year, indicating return of many of our members to civilian practice.

The State Society has undertaken many new activities during the past few months. The Louisiana Physicians Service, Inc., is now in full operation. News of this project has been carried in previous issues of the Journal and will be found in this issue also. The plan for medical service for veterans has been approved by our Executive Committee and submitted to the Veterans' Administration for confirmation. It is hoped that all details may be worked out for this service without further delay as many requests are being received from veterans interested in the plan. The president has appointed a committee, of which Dr. A. V. Friedrichs is Chairman, to make arrangements for a Council on Public Relations which will be a vital part of our future activity.

The importance of prompt payment of dues for continuation of the many activities of our organization can readily be understood and it is hoped that each member will realize the necessity of cooperation and feel his responsibility in these undertakings by remitting his dues promptly.

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PREPAYMENT SURGICAL PLAN EXPLAINED TO DOCTORS OF LOUISIANA

The Louisiana Physicians Service, Inc., started issuing prepayment surgical and obstetrical indemnity insurance contracts during the past month and within a short time it is expected some doctors will be approached by the insureds and a request made for surgical service as provided for in the policy certificate. Comments are made in this issue of the Journal to explain to the doctors exactly how the plan will work and also to request the doctors' participation in the plan.

Louisiana Physicians Service, Inc., is the result of efforts by the Louisiana State Medical Society through its Committee on Voluntary Prepayment Medical Service to give to the people of this state a voluntary prepaid medical benefit plan.

The Corporation was organized August 18, 1946 under the Laws of the State of Louisiana pertaining to the operation of a Mutual Industrial Insurance Company.

The company is under the supervision and control of the Commissioner of Insurance of the State of Louisiana and received its Certificate of Authority dated September 30, 1946 from Wade O. Martin, Jr., Secretary of State.

PARTICIPATING PHYSICIANS

Doctors of medicine licensed by the Louisiana State Board of Medical Examiners may sign an agreement with Louisiana Physicians Service, Inc.

The participating physician remains free to act in relation to patients in accordance with the custom in ordinary practice.

Louisiana Physicians Service does not contract to provide medical care nor may a participating physician be compelled to perform his services in any way differing from usual custom, except that the participating physician agrees to accept the fees in the Schedule of Benefits as full payment for services rendered under the Participating Physicians Agreement after determining with the subscriber that his yearly income, in the immediate preceding twelve months did not exceed $2,000, if single; or the combined income of the spouse and family group did not exceed $3,000.00.

The physician will make his usual charge to patients whose income is in excess of the amounts stated. Patients whose income is in excess of the brackets stated will pay that portion of the fee, if any, that remains after payment has been made by Louisiana Physicians Service, Inc., to the participating physician.

The subscriber and/or his dependents may select any doctor of medicine properly registered by a state board of medical examiners and payment will be made in accordance with the indemnity provided for in the Schedule of Benefits, provided the subscriber's surgical benefit certificate is in force at the date of service.

It should be noted that Louisiana Physicians Service, Inc., has no agreement with any but participating physicians.

PARTICIPATING PHYSICIANS AGREEMENT

The Participating Physicians Agreement is the contract between the participant and Louisiana Physicians Service. There are no dues, fees or costs of any type associated with becoming a participating physician. The physician may discontinue his agreement with the company on thirty days' notice as provided in the agreement. All the company wants is the assurance that the physicians of the state will cooperate with the prepayment medical care plan as established and sponsored by the Louisiana State Medical Society.

SUBSCRIBERS SURGICAL BENEFIT CERTIFICATE

I Benefits Provided

The term "surgical and other services," as used herein, is defined to mean any of the following services rendered while the certificate is in force, and not during a waiting period, by a doctor of medicine of the subscriber's choice, to the subscriber, or a dependent, or sponsored dependent covered by this certificate, except those items shown as exclusions under paragraph F.

Surgical Services

A. Surgical services (operative and cutting procedures for treatment of disease and injuries, and treatment of fractures and dislocations).

Obstetrical Services

B. Obstetrical services for abortion, miscarriage, ectopic pregnancy or delivery, but not until after the certificate has been in force for ten (10) consecutive months as a Family Group contract.

X-Ray Services

C. Diagnostic x-ray for surgical services not in excess of the value of Fifteen Dollars ($15.00) between the effective date of the certificate, and the first anniversary thereof, or during any succeeding twelve (12) month period.

Anesthesia

D. Anesthesia services not in excess of the value of Fifteen Dollars ($15.00), between the effective date of the certificate and the first anniversary thereof, or during any succeeding twelve (12) month period.

Laboratory

E. Laboratory services, but only when necessary for surgical diagnosis, and when not available through other insurance, and then not in excess of the value of Five Dollars ($5.00) for each surgical procedure not a part of a series of operations related to the same cause.

Benefits Not Provided

The following services or materials will not be provided under Louisiana Physicians Service Certificate.

Exclusions

F. "Surgical and other services," as used herein, does not include:

1. Hospital, dental or nursing services.
2. Medicines, drugs, appliances, materials, or supplies.
3. Services for industrial injuries or diseases, or services from any government agency, which are, or to the extent such services can be obtained by the subscriber without cost to him by compliance with laws enacted by a federal, state, municipal or other legislative body.
4. Services for self-inflicted injuries while sane or insane.
5. Benefits in excess of One Hundred Fifty Dollars ($150.00) between the effective date and the first anniversary of the certificate, or during any succeeding twelve (12) month period, for a series of related surgical or obstetrical procedures performed at the same time, or for different surgical or obstetrical procedures arising from the same medical cause.
6. Total benefits in excess of Three Hundred Dollars ($300.00) for all surgical and obstetrical services rendered under the certificate between the effective date and the first anniversary thereof, or during any succeeding twelve (12) month period.
7. Plastic operations for cosmetic or beautifying purposes.

Waiting Periods

The certificate will cover surgical or other services rendered in the following types of cases only after the prescribed waiting period has elapsed:

For maternity cases or conditions of pregnancy, ten (10) consecutive months from the effective date of the certificate as a Family Group contract.

For tonsillectomy, adenoidectomy, hemorrhoids, and hernia, three (3) consecutive months from the effective date of the certificate.

For known preexisting conditions (other than those covered by the three (3) month waiting period above), twelve (12) consecutive months from the effective date of the certificate.

PAYMENT OF FEES

Louisiana Physicians Service will make payment directly to the doctor of medicine who rendered service to a subscriber or dependent listed on subscriber's application, provided the doctor of medicine obtains from the subscriber an assignment of the benefits payable under the certificate. A prepared assignment is contained on the

Physicians Service Report and all the physician has to do is have the subscriber sign the assignment of fees payable.

IDENTIFICATION CARD

The applicant or employed subscriber always receives a Surgical Benefit identification card at the time of enrollment. He is impressed with the necessity of presenting this card to the doctor when service is requested. Unfortunately, some subscribers do not do so.

IT IS THEREFORE NECESSARY FOR THE DOCTOR OR THE DOCTOR'S OFFICE ASSISTANT TO ASK EACH PROSPECTIVE SURGICAL PATIENT IF HE OR SHE IS A MEMBER OF LOUISIANA PHYSICIANS SERVICE. We recommend that the doctor secure the following information from the card.

1. Name of subscriber (The person who signed the original application card for the Plan).
2. Address of subscriber.
3. The name of the patient.
4. The number of the certificate.
5. The group number of place of employment.

This permits prompt identification when the case is reported to our office, and, in turn, facilitates handling and payment to the participating physician.

The fact that a person presents an identification card does not necessarily mean he is entitled to surgical service. In order to assure payment of the benefits the doctor or the doctor's office assistant should write the home office or branch office or telephone the branch office listed on the identification card to determine if the patient is eligible for surgical service.

PHYSICIANS SERVICE REPORT

Upon completion of service to a subscriber or dependent listed on application to physician should complete the Physicians Service Report. The report is a simple one page blank that requires only a few minutes to complete. All of the claim blank "red tape" has been eliminated. The form has been designed for brevity and for convenience of the physician

The physician will be furnished with a number of these forms at the outset and additional forms may be obtained by writing Louisiana Physicians Service, Room 103, 1430 Tulane Avenue, New Orleans, Louisiana.

By the physician obtaining the assignment of benefits he is assured of receiving the fees payable under the Schedule of Benefits provided the subscriber is entitled to receive the services performed.

Participating Physicians should bear in mind that the fees established herein cover EXAMINATION, OPERATION and AFTER CARE of all SURGICAL conditions. Aftercare is defined as a reasonable postoperative follow-up period for the type of case in question (as established by experience in local surgical practice) and does not include medical treatment of/for non-surgical conditions which may occur coincidentally with surgery. Care and treatment of medical cases are not covered by this Plan at this time. Contingent on growth of the organization's reserves, the policies in the future are expected to cover all phases of medical service, including perhaps house calls.

SCHEDULE OF BENEFITS

The Schedule of Benefits of Louisiana Physicians Service, Inc., includes the more frequent surgical procedures. The Board of Directors recognizes that it would be futile to attempt to list each possible surgical operation.

The fees listed in the schedule are considered fair average fees for the patients UNDER the SPECIFIED INCOME limits and are accepted by the Participating Physician as COMPLETE PAYMENT FOR HIS SURGICAL SERVICES, to subscriber and dependents eligible for full service benefits. THESE FEES ARE NOT INTENDED TO REPRESENT WHAT THE SURGEON'S TOTAL CHARGE WILL BE OR SHOULD BE TO PATIENTS IN THE OVER INCOME GROUP, BUT ARE THE MAXIMUM REIMBURSEMENTS ALLOWED BY THIS PLAN. Therefore, it is extremely important that the patient and the doctor agree on the amount of any additional fee before services are rendered.

Services listed under "Obstetrics" are applicable only to Family Group Contracts.

The schedule fixes and limits the payment to be made by Louisiana Physicians Service, Inc., on account of surgical services covered by the Certificate, and payment shall in no case exceed the applicable rate fixed in the schedule.

Surgical procedures not specified in the Schedule of Benefits, but otherwise falling within the terms of the subscriber's Certificate, will be paid for at reasonable prevailing charges, which must be approved by Louisiana Physicians Service, Inc.

SCHEDULE OF BENEFITS FOR SURGICAL AND OBSTETRICAL SERVICES

ABDOMEN:

Appendectomy	$100.00
Cholecystectomy	150.00
Gastrectomy	150.00
Herniotomy (hernia), bilateral	100.00
Herniotomy, single	75.00
Paracentesis, abdominal	10.00
Other cutting through the abdominal wall for treatment or removal of organs in abdominal and pelvic cavities (unless otherwise specivied below)	100.00

ABSCESSES:

Carbuncle (massive) excision	$ 25.00
Furuncle, incision and drainage	5.00

AMPUTATIONS:

Arm, forearm, hand	$ 75.00
Coccyx, excision	50.00
Fingers or toes	25.00
Hip or shoulder	100.00
Thigh, leg	100.00
Ankle, foot	75.00

ANESTHESIA:

Major	$ 10.00
Major, over two hours	15.00
Minor	5.00

ARTERIES AND VEINS:

Varicose veins, excision or ligation	$ 50.00

BONES AND JOINTS:

Arthrodesis, large joints	$100.00
Arthrodesis, small joints	75.00
Arthroplasty, hip, knee, elbow	100.00
Arthrotomy	75.00
Bone Graft	100.00
Laminectomy	100.00
Spinal Fusions	100.00

BREAST:

Mammary gland, excision of tumor for biopsy	$ 25.00
Mammectomy, simple	75.00
Mammectomy, radical	150.00

CHEST:

Thoracoplasty	150.00
Bronchoscopy	50.00
Empyema, rib resection	75.00
Pneumothorax, initial induction	10.00
Refills	5.00
Maximum	25.00
Paracentesis	10.00
Phrenicotomy or phrenic avulsion	50.00

DEFORMITIES-CONGENITAL:

(Indemnity applies only to children under 18 years of age when covered by a Family Group Contract.)

Club foot	$100.00
Hairlip and cleft palate	100.00
Luxation of hip	100.00

DISLOCATIONS:

Reduction of temporo-mandibular	$ 10.00
Dislocation of the spine	100.00
Shoulder	40.00
Elbow, closed	35.00
Elbow, open	75.00
Fingers or toes	5.00
Hip	75.00
Knee	50.00
Ankle	40.00
Collarbone	25.00

EAR, NOSE AND THROAT:

Nasal Septum (sub-mucous resection	$ 75.00
Laryngoscopy: Removal of foreign body	50.00
Removal of growth	150.00
Esophagoscopy with foreign body extraction	75.00
Tonsillectomy and Adenoidectomy	35.00
Tracheotomy	50.00

Mastoid, simple	100.00
Mastoid, bilateral or radical	150.00
Antrostomy, window	50.00
Puncture eardrum	10.00
Puncture of antrum	5.00
Nasal polyp removal	15.00

EYE:

Foreign body removed from cornea	$ 5.00
Foreign body removed from inside eye or orbit	100.00
Primary suture of lid wounds	10.00
Iridectomy	60.00
Cataract, extraction	100.00
Cataract, needling	50.00
Detached retina operation	100.00
Removal of eyeball	100.00
Muscle operation	75.00
Operation for perforating wounds of the eyeball	100.00
Glaucoma	100.00
Chalazion	15.00
Dacryocystectomy	75.00
Dacryocystorhinostomy	100.00

FRACTURE:

Skull, operative	$100.00
Nasal bones	25.00
Maxilla	50.00
Mandible, unilateral	50.00
Mandible, bilateral	100.00
Malar	25.00
Clavicle, closed	25.00
Clavicle, open	50.00
Scapula	50.00
Ribs	5.00
Vertebra	100.00
Humerus, closed	50.00
Humerus, open and fixation	100.00
Radius or ulna	25.00
Radius and ulna, closed	75.00
Radius and ulna, open	100.00
Elbow, simple	50.00
Elbow, open reduction	100.00
Fingers	25.00
Pelvis	75.00
Femur	100.00
Knee cap	50.00
Tibia or tibia and fibula, closed	75.00
Tibia or tibia and fibula, open	100.00
Fibula, closed	25.00
Ankle, closed	50.00
Ankle, open	100.00
Toes, closed	25.00
Toes, open	50.00

GENITO-URINARY:

Cystoscopy — without radiography	$ 15.00
Cystoscopy — with radiography	25.00
Cystoscopy, operative	50.00
Urethrotomy, external	50.00
Hydrocele	50.00
Variocele	50.00
Nephrectomy	150.00
Orchidectomy	50.00
Prostatectomy (one or more stages)	150.00
Vasectomy	25.00
Removal of tumors or stones in ureter or bladder	100.00
Removal of tumors or stones in kidney, open operation	150.00
Circumcision, new born	5.00
Child to 12 years of age	10.00
Adult	15.00

GYNECOLOGY:

Operation strictly intra-vaginal or intra-uterine	$ 50.00
Hysterectomy	150.00
Fistula	50.00
Salpingectomy	100.00
Ovarian cyst	100.00
Cystocele or rectocele	50.00
Combined	75.00
Suspension of uterus	100.00

OBSTETRICS:

(Note: Applies only when subscriber and spouse are enrolled on Family Group Contract for ten (10) months or more.)

Delivery, normal, including pre-natal and post-natal care	$ 50.00
Cesarean section	150.00
Ectopic Pregnancy	100.00
Abortion or miscarriage, including curettage	50.00

RECTUM OR ANUS:

Operation on rectum or anus	$ 50.00
Hemorrhoids, external, thrombosed, incision	5.00
Resection of rectum	150.00
Hemorrhoidectomy	50.00

TENDON OPERATIONS:

Ruptured biceps, quadriceps, or tendo-Achilles	$100.00

Repair of other severed tendons...	50.00

THYROID:

Thyroidectomy	$150.00
Ligation of thyroid arteries not followed by thyroidectomy........	50.00

GENERAL SURGERY:

Skin cysts	$ 10.00
Tumors, excision (for biopsy)	25.00
Wounds, superficial, suturing.....	5.00
Wounds, lacerated, extensive......	25.00

THE RAGING FIRE

Over a long period of time, the medical profession has been confronted with the threat of having forced upon it Federal bureaucratic control of the distribution of medical care. The principle was accepted that this would be a continuing menace until the American people — 130,000,000 people—fully understood the implication of these moves and had become familiar with the aims, methods, policy and vast achievements of American Medicine under our system of the private distribution of medical care.

The American Medical Association, the Medical Societies, and other advocates of free enterprise have importantly aided in this vast and essential educational effort.

The menace of the centralized control of the distribution of medical care is a danger that might be likened to a raging forest fire.

It is through the efforts of the various Medical Societies that more than fifty prepayment medical care plans are in operation in nearly every state of the nation. The doctors throughout the nation have organized their own plans to provide medical care for the people of the nation at a cost far less to the individual than would be required as a weekly payroll deduction for administration by the federal government. This is your plan.

If you are interested in "putting out the fire that is raging now" rather than in building an edifice to house fire-fighting equipment to be used sometime, somewhere, for some fire;

If you are interested in participating in the actual solution of the difficult problems of the distribution of medical care; interested in avoiding political medicine and preserving the private practice of medicine in the United States—

Then, enroll as a participating physician in the Louisiana Physicians Service, Inc., to provide prepayment medical care for the people of this State. Louisiana State Medical Society is financing the company. All that is needed, now, is the physician's cooperation—enroll now.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Nov. 11.	Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
Nov. 13.	Woman's Auxiliary, Orleans Club, 3 p. m. Touro Infirmary Staff, 8 p. m.
Nov. 14.	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Nov. 15.	Executive Committee, Hotel Dieu, 8 p. m.
Nov. 18.	Hotel Dieu Staff, 8 p. m.
Nov. 19.	I. C. R. R. Hospital Staff, 12 noon. Charity Hospital Medical Staff, 8 p. m.
Nov. 20.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m. Charity Hospital Surgical Staff, 8 p. m. The Orleans Society of X-Ray Technicians, 7:30 p. m.
Nov. 22.	L. S. U. Faculty Club, 8 p. m.
Nov. 26.	Baptist Hospital Staff, 8 p. m.
Nov. 27.	French Hospital Staff, 8 p. m.
Nov. 28.	Clinico-pathologic Conference, Touro Infirmary, 12 noon. DePaul Sanitarium Staff, 8 p. m.
Nov. 29.	New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Dec. 2.	Board of Directors, Orleans Parish Medical Society, 8 p. m.
Dec. 3.	Eye, Ear, Nose and Throat Staff, 8 p. m. Orleans Parish Radiological Society, 7:30 p. m.
Dec. 4.	Clinico-pathologic Conference, Marine Hospital, 7:30 p. m. Mercy Hospital Staff, 8 p. m.
Dec. 5.	Executive Committee, Baptist Hospital, 8 p. m.
Dec. 6.	Ochsner Clinic Staff, 8:30 p. m.

Dr. Robert A. Katz attended the 25th anniversary meeting of the discovery of Insulin in Toronto, September 16-18. Dr. Katz presented a paper on Diethyl Ether in the Treatment of Ischemic Gangrene.

Dr. John T. Sanders attended the meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons at Hot Springs, Virginia, September 5-7.

WARNINGS FROM THE BETTER BUSINESS BUREAU

It is suggested that you get in touch with the Bureau if you are solicited by the following organizations:

Louisiana Parishes Safety Council, Inc. (J. E. Slaughter).

Western Tax Council, Chicago.

Navy Mothers Club.

The Club Women's Newspaper.

Make it a habit to check all solicitations or gifts by strangers with the Bureau.

CHANGES OF ADDRESS

Dr. Lucien C. Delery, 1708 Pere Marquette Bldg.
Dr. Bernard A. Goldman, 1510 Amelia Street.
Dr. Mercer G. Lynch, Ochsner Clinic.
Dr. Carl H. Rabin, 1510 Amelia Street.
Dr. Albert L. Braunstein, 206 Medical Arts Bldg.
Dr. Hardee Bethea, 1615 American Bank Bldg.
Dr. James S. Webb, 2908 Napoleon Avenue.
Dr. N. Leon Hart, 710 Pere Marquette Bldg.
Dr. John C. Suares, 319 Richards Bldg.
Dr. M. P. Boebinger, 4427 So. Robertson St.

WELCOME HOME

The following physicians were recently separated from military service:

Dr. Louis I. Tyler, Jr., 7215 So. Claiborne Ave.
Dr. Richard W. Vincent, 1615 American Bank Bldg.
Dr. Richard E. Robichaux, Raceland, La.

CALLED TO MILITARY SERVICE

Dr. Thomas G. Baffes was recently commissioned a first lieutenant in the Medical Corps of the Army of the United States. Lt. Baffes is stationed at the Veterans Administration, St. Cloud, Minn.

CHANGES IN MEMBERSHIP ROLLS

During the month of September the following changes took place in our membership rolls:

Two intern members (Drs. Arthur A. Calix and Herbert deN. Tucker) were transferred to active membership.

One active member (Dr. Daniel M. Kingsley) was transferred to inactive membership because of his temporary removal from New Orleans.

One active member (Dr. Ruth Winston Kope) was removed from membership because of her removal to St. Petersburg, Florida.

One intern (Dr. Paul L. Getzoff) reinstated to membership and transferred to that of active.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

RAPIDES PARISH MEDICAL SOCIETY

The Rapides Parish Medical Society held its regular meeting at the Hotel Bentley in Alexandria on Monday, October 7. The following program was presented: Dr. Chester A. Fresh spoke on psychosomatic problems, Dr. D. W. Oungst discussed the Veterans' Administration and Dr. O. J. Richardson spoke on the organization of visiting staff at Charity Hospital.

RESOLUTION OF TANGIPAHOA PARISH MEDICAL SOCIETY

We, the Tangipahoa Parish Medical Society, feel that certain facts should be called to the attention of the Louisiana State Medical Society;

A local patient, treated for two years for diabetes as proven by reports of a registered technician, had occasion to visit another physician. He was there informed that he did not have diabetes, but that he had a cancer with only a short time to live.

Feeling that he had been handled in an improper and careless manner, he returned to the local doctor who had treated him, accused him as indicated, then murdered the doctor and committed suicide. Notes found on the patient's person and made public by the coroner revealed this act as a clearly premeditated murder.

We, the doctors of the Tangipahoa Parish Medical Society, wish to express our regret that we have doctors in the field of medicine who do not respect medical ethics and take this means to register our disapproval as this is not the only instance that has come to our attention of a lack of ethics by our colleagues.

Collins P. Lipscomb, M.D.
Secretary

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

A meeting of the Southern Section of the organization was held on Friday, September 27 at the Louisiana State University School of Medicine. The following program was presented:

1. Hematologic Effects of Pteroyl Glutamic Acid in Man. Grace A. Goldsmith (introduced by J. H. Musser), Department of Medicine, Tulane University School of Medicine.
2. Experiments Dealing with the Life History of the Hookworm *Bunostonum Phlebotomum* Parasitic in Calves. Roy L. Mayhew, College of Agriculture, Louisiana State University, Baton Rouge.
3. Differences in Culturability, Infectivity and Pathogenicity of Human Strains of *Endamoeba Histolytica.* Ernest Carroll Faust, Department of Tropical Medicine, Tulane University School of Medicine.
4. An Easy and Accurate Method for Determination of Hemoglobin in Blood, Based upon Its Iron Content from One Drop of Finger-tip Blood. J. T. Peters, Department of Medicine, Louisiana State University School of Medicine.
5. Blood Volume Studies in Congenital Heart Diseases. Woodrow Nelson, H. S. Mayerson, John H. Clark, and Champ Lyons. Departments of Physiology and Surgery, Tulane University School of Medicine.
6. Radiosodium Tracer Studies in Congestive Heart Failure. Paul Reaser and George Burch, Department of Medicine, Tulane University School of Medicine.

PUBLIC HEALTH TRAINING CENTER

The first of October there was opened a Public Health Training Center in the City of New Orleans which is jointly supported by the City and State and which is being given financial assistance by the Commonwealth Fund of New York. The Center will be under the direction of Dr. Ben Friedman who conducted the training center that was supported by the State Board of Health at Franklin, Louisiana, prior to his going into the Army.

The present training center will be a very much more elaborate institution. There will be doctors, trained nurses and sanitarians, as well as health, welfare and social workers. The personnel will be trained both for the City and State Department of Health.

Dr. Friedman was on the staff of General Eisenhower during the war and amongst other honors he received two decorations from the Norwegian Government.

NEWS ITEMS

Dr. Ralph Platou, Professor of Pediatrics at Tulane, addressed the Southern Tuberculosis Conference at Jacksonville, Florida, on "The Management of Primary Tuberculosis in Children."

Dr. Maurice Campagna, of the medical department of Tulane, gave an address on the important subject "Interesting the General Practitioner in Tuberculosis."

Dr. Julius Lane Wilson, president of the State Tuberculosis Association, and Dr. John M. Whitney also were in attendance at this meeting.

Dr. Michael DeBakey of the Department of Surgery, Tulane University, is one of eight members of the new committee called the Medical Advisory Committee, appointed by Secretary of War, Robert P. Patterson.

Miss Mary Louise Marshall, Librarian of the Orleans Parish Medical Society of the Tulane University Medical Library, was one of six speakers who addressed the Association of Honorary Consultants to the Army Medical Library.

The National Gastroenterological Association announces it Annual Cash Prize Award Contest for 1947. One hundred dollars and a Certificate of Merit will be given for the best unpublished contribution on gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy.

The second Inter-American Congress of Radiology will be held in Havana November 17-22. Reservations may be made at the Sevilla-Biltmore. Dr. Leon J. Menville is one of six of the general committee for the United States.

The American Academy of Allergy will hold its annual convention at Hotel Pennsylvania, New

York City, November 25-27. All physicians are cordially invited to attend the sessions as guests of the Academy.

Dr. Myron Wegman, Head of the Department of Pediatrics of the Louisiana State University School of Medicine, attended the Fourth Postgraduate Conference in Pediatrics to be held in Galveston, October 28-November 2. Dr. Wegman addressed the Conference on the topics:

1. Scope diarrhea problems
2. Factors influencing growth and development
3. Common immunization procedures.

Dr. Russell L. Holman, Head of the Department of Pathology and Bacteriology of the Louisiana State University School of Medicine, attended the meeting of the Southern Medical Association in Miami, Florida. On November 5, 1946, he addressed the Section on Medical Education and Hospital Training of Southern Medical Association on "The Integration of the Teaching of the Basic Medical Sciences With The Hospital Services."

Dr. Lyon K. Loomis, Instructor of Orthopedics, also attended the meeting of the Southern Medical Association. His address was on: "Treatment of Simple Oblique and Simple Spiral Fracture of the Shaft of the Tibia."

A splendid opportunity exists for a young man to obtain training in one of the outstanding European hospitals, namely the American Hospital in Paris. Applications for such a position are now being accepted. To the man who may be interested in this position he should take the matter up with Dr. John Archinard of New Orleans.

The American Foundation of Tropical Medicine will establish in Liberia an institute for research in tropical medicine. This undertaking has been made possible through a large gift from Mr. Firestone of the tire and rubber company.

SCIENTIFIC EXHIBIT

Centennial Session—American Medical Association

At the Centennial Session of the American Medical Association to be held in Atlantic City, June 9-13, 1947, the Scientific Exhibit will include both the history of medicine during the past Century and the latest developments of medical science.

Application blanks for space are now available. All applicants must fill out the regular form. Applications close on January 13, 1947, after which time the Committee on Scientific Exhibit will make its decision and notify the applicants.

Application blanks for space should be procured as soon as possible. They are available from The Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

THE NATIONAL CANCER FOUNDATION

This organization will put on its first annual appeal for funds during the month of November. The campaign will be carried on throughout the country. Part of the money obtained from the campaign will be utilized in building hospitals for advanced cancer patients.

CONFERENCE ON GASTRIC CANCER

There will be held December 5-6 at the Billings Hospital, University of Chicago, under the chairmanship of Surgeon General Parran of the United States Public Health Service, a conference on gastric cancer. This is the third such conference that has been held since the original one in 1940. The importance of gastric cancer may be illustrated by the fact that cancer of the gastro-intestinal tract causes 45 per cent of all cancer deaths in this country.

A distinguished group of clinicians, surgeons, pathologists, radiologists, and others will participate in the program.

DALLAS SOUTHERN CLINICAL SOCIETY

The Dallas Southern Clinical Society will hold a postgraduate medical conference November 18-21. These courses are in medicine, surgery, obstetrics and pediatrics.

CAMPAIGN TO COMBAT HEART DISEASE

The American Heart Association announces initiation of a nation-wide program on public education of heart disease. The organization points out that heart disease now kills more than any other disease. In fact the number of deaths due to diseases of the heart is greater than the total of the next five leading causes of death.

The Heart Association will have the support and cooperation of many well known national organizations such as the American Academy of Pediatrics, American College of Physicians and the American Public Health Association.

In pursuance with their purpose of public education there will be held a National Heart Week, beginning February 9, 1947.

The Metropolitan Life Insurance Company is an extremely active participant in this educational campaign. The company has prepared three monographs on heart disease which will be distributed to physicians throughout the country. The monographs are extremely well gotten up and are of interest not only to the physician but primarily to the lay individual. These are prepared for distribution to doctors with the hope that they will place them in their offices or make available to such groups as service clubs, parent-teacher associations and other civic organizations.

NEW YORK RHEUMATISM ASSOCIATION

There will be a scientific meeting of the New York Rheumatism Association on Friday evening, November 22, 1946 at 8:15 p. m., at the Cornell University Medical College Auditorium.

TABLE OF METRIC DOSES WITH APPROXIMATE APOTHECARY EQUIVALENTS

Liquid Measure

Metric	Approximate Apothecary Equivalents
1000 cc.	1 quart
750 cc.	1½ pints
500 cc.	1 pint
250 cc.	8 fluidounces
200 cc.	7 fluidounces
100 cc.	3½ fluidounces
50 cc.	1¾ fluidounces
30 cc.	1 fluidounce
15 cc.	4 fluidrachms
10 cc.	2½ fluidrachms
8 cc.	2 fluidrachms
5 cc.	1¼ fluidrachms
4 cc.	1 fluidrachm
3 cc.	45 minims
2 cc.	30 minims
1 cc.	15 minims
0.75 cc.	12 minims
0.6 cc.	10 minims
0.5 cc.	8 minims
0.3 cc.	5 minims
0.25 cc.	4 minims
0.2 cc.	3 minims
0.1 cc.	1½ minims
0.06 cc.	1 minim
0.05 cc.	¾ minim
0.03 cc.	½ minim

Weight

Metric	Approximate Apothecary Equivalents
30 Gm.	1 ounce
15 Gm.	4 drachms
10 Gm.	2½ drachms
7.5 Gm.	2 drachms
6 Gm.	90 grains
5 Gm.	75 grains
4 Gm.	60 grains (1 drachm)
3 Gm.	45 grains
2 Gm.	30 grains (½ drachm)
1.5 Gm.	22 grains
1 Gm.	15 grains
0.75 Gm.	12 grains
0.6 Gm.	10 grains
0.5 Gm.	7½ grains
0.4 Gm.	6 grains
0.3 Gm.	5 grains
0.25 Gm.	4 grains
0.2 Gm.	3 grains
0.15 Gm.	2½ grains
0.12 Gm.	2 grains
0.1 Gm.	1½ grains
75 mg.	1¼ grains
60 mg.	1 grain
50 mg.	¾ grain
40 mg.	2/3 grain
30 mg.	½ grain
25 mg.	3/8 grain
20 mg.	1/3 grain
15 mg.	¼ grain
12 mg.	1/5 grain
10 mg.	1/6 grain
8 mg.	1/8 grain
6 mg.	1/10 grain
5 mg.	1/12 grain
4 mg.	1/15 grain
3 mg.	1/20 grain
2 mg.	1/30 grain
1.5 mg.	1/40 grain
1.2 mg.	1/50 grain
1 mg.	1/60 grain
0.8 mg.	1/80 grain
0.6 mg.	1/100 grain
0.5 mg.	1/120 grain
0.4 mg.	1/150 grain
0.3 mg.	1/200 grain
0.25 mg.	1/250 grain
0.2 mg.	1/300 grain
0.15 mg.	1/400 grain
0.12 mg.	1/500 grain
0.1 mg.	1/600 grain

The above *approximate* dose equivalents have been adopted by the latest Pharmacœpia, National Formulary, and New Non-official Remedies, and these dose equivalents have the approval of the Federal Food and Drug Administration.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending September 22 there occurred in the city of New Orleans 150 deaths which were divided 89 white, 61 colored; 20 of the deaths were in children under one year of age divided equally between the two races. For the following week which came to an end September 14 there was a rather sharp drop in the deaths taking place in the city of New Orleans; 89 of the people that died were white individuals, 33 colored. Only nine were children under one year of age and eight of them were white children. The succeeding week which closed September 21 found 108 New Orleanians expiring, of whom 57 were white and 51 were colored. Seventeen of these deaths were infant deaths with the figures for the previous week practically reversed between the two races. For the week ending September 28 the 122 deaths in the city were divided 80 white, 42 colored. Seventeen of the deaths were in children under one year of age and this week 13 of these children were white and only four negro. The mortality rate in New Orleans still remains well below that of the previous three year average.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending September 7, showed the following reportable diseases in numbers greater than 10: 72 cases of malaria, 28 of pulmonary tuberculosis, 17 of unclassified pneumonia, 16 of poliomyelitis and 12 of whooping cough. The poliomyelitis cases were pretty well scattered throughout the state. No one parish reported more than two cases except East Baton Rouge with four. For the following week ending September 14, there was again a remarkable diminution in the number of reportable cases. Pulmonary tuberculosis led with 25, followed by poliomyelitis with 21 and typhus fever with 12 instances. Again this week the poliomyelitis cases came from all over the state, but Orleans Parish reported seven and East Baton Rouge four. For the week which closed September 21, malaria was at the head of the list with 54 cases, all contracted within the continent of the United States. Next came poliomyelitis with 20 cases, followed by unclassified pneumonia with 16, septic sore throat with 15, pulmonary tuberculosis 14 and typhoid fever 10. Of the large number of poliomyelitis cases being reported, Orleans Parish and Caddo were the only parishes that had more than two cases. The 10 cases of typhoid fever occurred in 10 separate and distinct parishes. For the week which ended September 28, for the first time, in listing the number of cases in the state of various reportable diseases, pulmonary tuberculosis was the only disease which occurred in number greater than 10, there being 38 patients listed with this disease this week. The epidemic of poliomyelitis seemed to be on the wane with only five cases reported.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending August 31, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	68	410	478
Gonorrhea	1382	11875	13257
Granuloma Inguinale	28	191	219
Lymphopathia Venereum	5	78	83
Syphilis	1799	11961	13760

LAKESHORE HOSPITAL

On June 1, 1946 Lakeshore Hospital located at West End Boulevard and Lakeshore Drive opened. This Hospital was created by the New Orleans Medical Foundation for the purpose of providing sorely needed additional hospital beds for the City of New Orleans and its vicinity. The Hospital is a non-profit institution operated by the New Orleans Medical Foundation, the membership of which consists entirely of veteran medical officers of the armed forces who served in World War II.

Even though the Lakeshore Hospital is operated by an organization composed entirely of veteran medical officers this is not to be misconstrued to mean that membership on the staff of the Hospital is limited to returning medical officers. The actual operation of the Hospital is controlled by a board of directors composed of the members of the executive committee of the New Orleans Medical Foundation plus representatives of other medical groups and hospitals in the City of New Orleans, as follows: the president and the president-elect of the Orleans Parish Medical Society, the president of the New Orleans Hospital Council; and one member of the board of directors of each of the following Hospitals: Touro Infirmary, Hotel Dieu, Mercy Hospital, and Southern Baptist Hospital. In addition to these there are two members at large who were selected by the above members of the Board of Directors.

Even though membership of the staff is open to all doctors licensed in the State of Louisiana who are members of the Orleans Parish Medical Society, applications for membership to the staff are carefully reviewed by the medical committee of the Board of Directors prior to granting the use of the facilities of the Hospital. In effect, this creates an open staff for Lakeshore Hospital which is under the rigid control of the medical committee in order to maintain the existing standards of hospital care in the City of New Orleans.

Lakeshore Hospital is a general hospital fully equipped to take care of all types of medical and surgical cases and has excellent laboratory, x-ray, surgical, and obstetrical facilities. There is also a competent resident staff of doctors capable of assisting the members of the staff in the care of their patients. The Hospital is not equipped to handle neuro-psychiatric problems, terminal care cases, alcoholic cases, or drug addiction cases. If these conditions co-exist with other medical or surgical conditions care can be provided for these types of cases only to the extent where the medical or surgical condition has been brought under control.

The nursing personnel and staff of Lakeshore Hospital is of the highest calibre and experience has demonstrated that in the performance of their duties they are rendering the highest type of nursing service. Miss Imogene Yarbrough is Director of Nurses. The doctors who have had patients at the Hospital have only the highest commendation for the quality of nursing which she has been able to provide. Those on the Board of Directors who have worked with Miss Yarbrough can only express their appreciation and thanks to her interest and devotion in developing such an excellent nursing staff.

The services though not straight services in the strict sense of the word are divided for the purpose of organization and operation into the following categories: internal medicine, general surgery, orthopedic surgery, obstetrics and gynecology, ophthalmology, and otolaryngology. The staff is similarly organized. The present bed capacity is 150 beds with existing renovations provided for an expansion to 175 beds in the immediate future. Other existing buildings are to be made available should additional beds be needed. The anticipated maximum bed capacity can be expanded to 325 beds. The diagnostic and pathological laboratories are fully equipped and adequately staffed. They are capable of performing all laboratory and diagnostic procedures. The direction of the laboratory is under the supervision of Dr. Friedrichs. It is anticipated that in the very near future a full time medical director of the laboratory will be available at the Hospital for consultation with the doctors and supervision of the laboratory procedures.

In addition to the regular laboratory service is proud to announce that it has the only fully equipped Tropical Diseases Laboratory connected with a private hospital in this section of the country. This Laboratory is under the direction of Dr. Joseph D'Antoni and is available to all members of the staff for diagnostic and consultation services.

The X-Ray Department is under the direction of Dr. J. N. Ané and Dr. H. F. Inderlied. These two doctors will provide 24-hour coverage for the x-ray service. They are available for consultation with the staff doctors at all times. The x-ray equipment is of the best quality and is capable of recording all types of x-ray examinations.

The technical assistants in all laboratories as well as the heads of all departments such as the dietitian and the pharmacist, are registered as certified in their respective fields. Mrs. C. L. Garris arrived at the Hospital on September 13, 1946 and immediately began her work as dietitian for the Institution. In addition, Mrs. Shelby McCaffrey, who is in charge of the Medical Records Library, is a certified Medical Record Librarian. The quality of her work is well known to doctors and her presence assures the Hospital that the records of the patients will be well supervised and duly recorded.

The surgical facilities at Lakeshore Hospital are second to none as existing in the City of New Orleans. The direction of the operating room is under Miss Margaret Bussey who prior to assuming her duties at Lakeshore Hospital was supervisor of the operating room of Southern Baptist Hospital and the 64th General Hospital. The equipment of the operating suite is more than adequate to enable any and all types of surgical procedures to be performed. There are five large, well lighted, well ventilated operating rooms which provide sufficient space to maintain a very heavy daily operating schedule.

The obstetrical wing opened on September 15. The nursery of this service has a capacity at present of 30 bassinets with two incubators. There are four labor rooms and two delivery rooms. The predelivery wing is separate from the post-natal building. Contrary to existing policies of the other hospitals in New Orleans Lakeshore Hospital has not as yet felt that it is necessary to limit post-delivery stay of the patients. It is hoped that if it is so desired all post-partum cases will be able to remain in the Hospital for at least ten days, perhaps in certain cases for a longer period of time.

At this time the Board of Directors of Lakeshore Hospital would like to announce that Dr. W. P. Gardner has been secured as Director of the Hospital. Dr. Gardner assumed his duties as of Monday, October 15. Prior to his arrival Mr. Paul Rutledge, business manager, has been filling the dual role of being both Director and Business Manager. During this period his industry and leadership have made him most successful and it is felt that the arrival of Dr. Gardner will be welcomed by Mr. Rutledge, so that Mr. Rutledge may resume his original position as business manager of the Hospital.

Lakeshore Hospital was opened to help meet the acute bed shortage in New Orleans. The waiting lists for admission to other hospitals in the City continue, indicating that further expansion of this Hospital may be necessary more completely to relieve this emergency. Looking beyond the emergency it is conceivable that the expected increase in population in New Orleans as a result of the shifting population during the War will represent to a great extent a permanent increase in population. In addition to this expanding industry and business will produce a still further increase in permanent population. Present time investigation has shown that additional permanent hospital beds should be added to those already existing in the City. The New Orleans Medical Foundation is cognizant of these conditions and is carefully investigating possibilities for solving this problem, because the members of this Foundation feel that the Foundation can fill a definite need in the medical program not only of the City of New Orleans but also of the Southern States. The ways and means of providing an increase in the permanent beds are being investigated as well as other problems pertaining to the medical care of the citizens of this vicinity. It is hoped that the Foundation can be not only of moral but also of material benefit in helping to solve some of these problems. With the cooperation of the doctors, and by cooperating with the doctors and other hospitals in the City, this Foundation can be of great assistance in making New Orleans a medical center of the South.

ALFRED B. LONGACRE, M. D.
Chairman Medical Committee.

BOOK REVIEWS

Hypnoanalysis: By Lewis R. Wolberg, New York, Grune & Stratton, 1945. Pp. 342.

There is an urgent need in psychiatry for an abbreviated form of therapy applicable to patients who are unable to avail themselves of prolonged treatment. Existent therapies include suggestion, persuasion, guidance, confession and mental ventilation, reassurance, re-education, desensitization and the correction of remediable elements in the patient's environment that have a destructive effect on his adjustment.

While such therapies are superficial, they are justified because they often eliminate sources of stress and favorably alter behavior that brings the individual into conflict with others. They may also provide a medium for the most effective operation of whatever spontaneous forces of mental health remain and establish the beginnings of more profound psychic change.

Hypnoanalysis has emerged as a procedure contributing speed and directness in therapy. Dr. Wolberg has written the most exhaustive available account of the theory and practice of hypnoanalysis and he has elaborated on the dynamics, indication, limitations, and technic of the method, evolving new experimental procedures. He believes hypnoanalysis has definite values, but will not move mountains and that failures occur as they do with other therapies but he also believes persons respond to hypnoanalysis when other psychotherapies have failed.

With true scientific spirit, the author states "hypnoanalysis holds promise of becoming an invaluable adjunct to psychoanalysis, but it must be explored more thoroughly before its scientific worth can be fully evaluated." The present volume is offered as a contribution in that direction and it is an extraordinarily valuable and important piece of work because human relations, human behavior and human reactions to the desire for security and happiness and the struggle for existence constitute the twentieth century challenge to psychiatry.

C. P. MAY, M. D.

Manson's Tropical Diseases. A Manual of the Diseases of Warm Climates: Ed. By Philip H. Manson-Bahr, C. M. G., D. S. O., M. A., M. D., D. T. M. and H. Cantab, FRCP, London, 12th Ed., Baltimore, Williams and Wilkins, 1945. Pp. 1068, por. illus. Price, $12.00.

The early editions of *Manson's Tropical Diseases* enjoyed almost a monopoly so far as English publications were concerned. Now, however, the situation is different and the twelfth edition comes in competition with several works published since the eleventh edition appeared in 1940. This work differs from some of the newer volumes in that it is the work of a single author whereas several of the other recent publications have been prepared by specialists in the different fields. This edition consists of 840 pages devoted to individual diseases and a few chapters on general subjects, followed by approximately 150 pages of medical protozoology in which the author includes medical helminthology and medical entomology. The latter is followed by approximately 20 pages of laboratory methods. A somewhat unusual feature is the table of drugs for the treatment of tropical diseases. In the preface the author states that many chapters have been rewritten. As a result many of the newer observations have been included. The work has been prepared particularly from the point of view of the clinician with less attention, but perhaps sufficient, to public health and preventive medicine aspects. In comprehensiveness the work stands between the newer concise manuals that have appeared in England and the United States, and the very extensive work of Stitt as revised by Strong. A few points of special interest may be mentioned: The difficulties of absolute differentiation of the cholera vibria from similar organisms are very clearly set forth. The author is impressed by the value of cholera vaccination in the control of the disease, and even is favorably impressed by oral vaccine. Under the treatment of leprosy chaulmoogra oil and its derivatives are given much space but no mention is made of promin, which has attracted so much attention in the United States in recent years. The author takes a very optimistic view of the results of attempts to control leprosy in the Philippines. Not all students of the subject will agree. Opium in full doses is believed by the author to have special virtue in phagedenic processes of tropical ulcers. The author prefers the term Bilharziasis for the parasitic infestation that, in recent years, is more often called Schistosomiasis. We are told that in Egypt mass treatment of cases is being practiced as a prophylactic measure. A few statements may need revision when the next edition is published—thus on page 242 we find the statement that the virus of Rocky Mountain Fever has not been found in small mamals which is correct, but in Table IV facing page 244, vertebrate reservoirs are mentioned which so far as present knowledge goes is incorrect. On page 248 under geographical distribution of Q Fever the statement is made that it is apparently widespread in America, without making clear that this refers to the distribution of the virus, not of the disease in man. So far as information is available, aside from laboratory infections, but a single outbreak in man has occurred in the United States. In the differentation of the plague organism from that of *Pseudo-tuberculosis rodentium* it is mentioned that rats are instantly killed by *B. pestis.* It is, of course, true that they die in a few days, but instantly is a little misleading. Penicillin is not mentioned under the treatment of yaws. There is no mention of the importance of tracheotomy in dealing with one of the more serious complications of leprosy. In the discussion of general effects of ankylostomiasis it is reported that there may be a predisposition to larceny and other crimes. In general the work is to be regarded as satisfactory and should prove very useful.

G. M. McCoy, M. D.

Narcotics and Drug Addiction: By Erich Hesse, M. D., New York Philosophical Library, Inc., 1946. pp. 219. Price, $3.75.

In this book the author has attempted to cover a wide field extending even beyond that commonly suggested by the title. In addition to the opiates, cocaine, cannabis and so on, which are included under the general heading of narcotics, consideration is also given to alcohol, tobacco and the purine drugs which are classified as stimulants, a rather broad and arbitrary use of the term. On the other hand, over indulgence in soporifics such as the barbituric acid compounds is treated very briefly. Some of the chapters most interesting to the reviewer concern habituations to less well known drugs such as mescaline, kava-kava, certain toadstools, and betel. In the treatment of each drug attention is given to its source, its method of preparation for use, its physical and chemical properties, an outline of its pharmacologic action, an estimate of the extent of its use, and its sociologic importance as a habit forming or addicting drug.

The author's definitions of habituation and addiction do not conform with those in use in this country at the present time, but this is of minor importance since his meaning of the terms is made clear. The content suffers to some extent in translation from the German by the use of unusual terminology and in some cases idiomatic construction. Probably a more serious criticism from the viewpoint of the interest of the American reader is that discussions of the extent of the use of various drugs and laws controlling drug traffic refer to Europe and particularly to Germany with only incidental reference to conditions in this country. Also bibliographic references are drawn chiefly from the German literature.

Although a comprehensive treatment of the pharmacology of each drug is unnecessary in a book of this type numerous statements are made and impressions given to which exception should be taken. In discussing the chronic effects of alcohol no reference is made to the part played by avitaminosis. The impression is given that cirrhosis of the liver may be produced by alcohol in experimental animals. References to the chemistry of the active principle of hashish indicate unfamiliarity with recent developments in this field. Also in numerous instances a positive stand is taken where the scientific evidence, is at best, controversial.

As stated in the preface, the purpose of the book is in part to combat drug addiction and habituation by the dissemination of knowledge concerning the dangers involved. In pursuit of this worthy purpose the author frequently gives greater credence to evidence of the deleterious effects of the drugs rather than to contrary reports when the literature on the subject is controversial. Although the reader will find much of interest it must be recognized that a thesis rather than a purely scientific consideration of the subject has been presented.

Ralph G. Smith, M. D.

Current Therapies of Personality Disorders: By Bernard Glueck, M. D. New York, Grune & Stratton, 1946. Pp. 296.

The results of the proceedings of the thirty-fourth annual meeting of the American Psychopathological Association, are available now in this volume to guide physicians in management and treatment of personality disorders. Twenty experts present a series of timely papers devoted to the modern psychopathic hospital, psychochemical technics, phychiatric guidance and rehabilitation. There is invaluable material for neuropsychiatrists that will help to rid personality disorders of the mystery clouding their proper medical recognition. You will come from reading this book with an enlarged conception of the human organism as an almost infinite complexity of action and reaction between structure, normal and abnormal, and function, orderly and disordered. It can be recommended to those who desire a comprehensive survey of the subject. It should present considerable food for thought to therapists in the field of human personality and human relations.

C. P. May, M. D.

Preoperative and Postoperative Treatment: Edited by Lt. Col. Robert L. Mason, M. C., A. U. S., and Harold A. Zintel, M. D. 2nd ed. Philadelphia, W. B. Saunders & Co. 1946. Pp. 584. Price $7.00.

This volume fills a real need. It should be on the shelf of every hospital library. It should be a welcome addition to the reference library of surgeons. It is an invaluable reference book for the resident staff.

Each chapter in this volume gives evidence of careful preparation. The material is as near the current literature as is possible for a printed volume to be. It is not often that one can say without hesitation — this is a volume which deserves a place in any man's library.

Isidore Cohn.

Textbook of Gynecology: By Arthur Hale Curtis, M. D. 5th ed. Philadelphia, W. B. Saunders, 1946. Illus. Pp. 755. Price $8.00.

This new edition has 455 illustrations and 755 pages. It has some new additions on almost every gynecologic subject, and numerous revisions. The new additions to the chapter on Other Tumors of the Uterus are nicely presented and well illustrated with photographic plates. The principal revisions noted are in endocrinopathic disturbances and ovarian tumors.

The illustrations and material from the fourth edition have been used and 54 new illustrations added. The general plan of presentation is the same.

This new book is an improvement over the fourth edition and is an excellent reference book for clinicians and students. It is very well written and illustrated, and presents recent trends in physiology, pathology and treatment in gynecologic disease.

Lee H. Lorenzen, M. D.

Bibliography of Infantile Paralysis, 1789-1944, with selected abstracts and annotations, prepared under the direction of the National Foundation for Infantile Paralysis, Inc.: Edited by Morris Fishbein, M. D., comp. by Ludvig Hektoen, M. D. and E. M. Salmonsen. Philadelphia, J. B. Lippincott, 1946. Pp. 672. Price $15.00.

Of widespread interest among neuropsychiatrists and orthopedists, should be this bibliography with abstracts from the literature, on infantile paralysis. It includes a comprehensive list of periodicals covering the clinical and investigative work on the subject and its phases since its description by Underwood in 1789.

The elements are arranged chronologically and access to the various aspects of the condition is facilitated by an extensive, complete and thorough index covering the references and articles by number. This method of arrangement has been found the most convenient of any of the various technics that are used in the preparation for publication of material of this kind. A vast amount of valuable data is thus made available.

C. P. May, M. D.

PUBLICATIONS RECEIVED

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INFECTIOUS HEPATITIS IN CHILDREN

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The high incidence of acute liver disease in the armed forces of the United States during World War II has stimulated marked interest in this subject. The outbreak of jaundice of mysterious type in the training camps in 1942, later demonstrated to be due to homologous serum was followed by epidemics of acute infectious hepatitis, especially in the Mediterranean theater of operations.[1] The establishment of the Hepatitis Commission, with small teams of research workers, resulted in the clarification of many details concerning these infections. The size of the clinical problem is evident in the report of one group from the Mediterranean theater which conducted a study of 1172 cases of hepatitis, in addition to special surveys on several thousand others. Two of these investigators had previously made extensive clinical and laboratory studies of 2000 trainees who had received yellow fever vaccine, of whom 700 were hospitalized with jaundice.[1]

Prior to these investigations, a considerable amount of confusion existed, despite a few reports which were pointing toward the present conception of acute hepatitis. The severe hepatitis due to *Leptospira icterohaemorrhagiae* had been differentiated by Weil in 1886 and shown to be of spirochetal etiology by Japanese investigators[2] in 1914. Homologous serum jaundice had been described by Findlay and MacCallum[3] in 1937 but was not generally recognized. Acute catarrhal jaundice was still considered to be different from acute infectious hepatitis, and to represent an ascending cholangitis, with jaundice resulting from mucus obstruction of the bile ducts, although Rich[4] and others had presented evidence that "catarrhal jaundice" was due to hepatitis of systemic origin and Blumer[5] had believed catarrhal jaundice to be the sporadic form of epidemic or infectious hepatitis. The frequency of residual liver damage after infectious hepatitis and the important role of dietary factors in preventing liver damage were not generally known.

Extensive investigations of homologous serum hepatitis and infectious hepatitis[6, 7] have established a number of salient facts concerning their etiologic agents, despite the failure of attempts to isolate, identify or grow causative organisms. The agents of both diseases are filterable viruses, are resistant to heat at 56 degrees C, survive for long periods of time at room temperature and in frozen feces or urine, survive exposure to 1:2000 merthiolate and are not affected by the ordinary methods of chlorination. Animal inoculations are unsuccessful, lesions are not produced in the chick embryo and inclusion bodies have not been found. They produce similar manifestations in humans, and pathologic changes of inflammation and necrosis of liver cells without involvement of the larger bile ducts are identical. Both agents are present in the blood at some time during the respective disease. The fatality rates are about the same (0.2 to 0.3 per cent) and fatal outcome in each group is preceded by marked evidences of central nervous system involvement.

Certain differences are noted. In infectious hepatitis, the incubation period is usually less than 40 days, while in serum hepatitis it is 60 to 150 days. The onset of homologous serum hepatitis is usually afebrile and the temperature usually does not exceed 100 degrees, whereas in infectious hepatitis, it usually exceeds 100 degrees F. Contact cases are rare in serum hepatitis, more common in infectious hepatitis. Neefe, Stokes and Gellis[7] present evidence that each disease produces homologous immunity, but not a cross immunity. Transmission in serum hepatitis is effected by injection of infected blood or serum, as in transfusions and other intravenous medications, or material containing serum as a stabilizing agent, as happened in the instance of yellow fever vaccine; infectious hepatitis is transmitted largely by ingestion of materials contaminated with feces.

The latter point was established by general observations that epidemics of the disease are more frequent under crowded, unsanitary conditions, and specifically by the epidemiologic studies of Neefe and Stokes[8] during an epidemic which occurred in a summer camp near Philadelphia in 1944, during which 350 (61 per cent) of a total of 572 boys, girls and adult attendants developed hepatitis. Epidemiologic observations and transmission experiments in human volunteers provided evidence that the causative agent was not transmitted by air, fomites, biting insects, flies, food, milk or water from the lake nor directly from infected persons, but was transmitted by water from a well which contained a transmissible agent which was apparently the same as that excreted in the feces of infected persons.

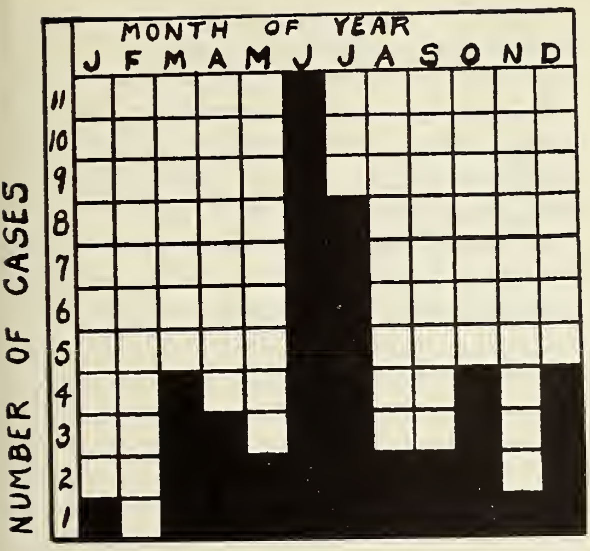
INCIDENCE OF HEPATITIS

A number of reports have indicated a marked increase in the incidence of virus gastroenteritis in the civilian population of the United States during the war years. In our area of North Louisiana this has been paralleled, to a lesser degree, by an apparent increased incidence of acute hepatitis. Our Clinic records show only one child with the disease during the ten years prior to 1941, with an incidence of 41 since that time (table 1). The increased incidence has been particularly noted during the past three years, with 16, 6 and 16 cases occurring during 1944, 1945 and 1946, respectively. In several areas, the disease appears to be endemic. In the town of Mansfield, a parish seat of about 5,000 population situated 40 miles south of Shreveport, ten to twelve children between the ages of one and ten years have had the disease since 1944, only one of whom is included in our Clinic series. Two other towns, Arcadia, La., and Atlanta, Texas, are known to have had four to six cases each. At the Shreveport Charity Hospital during the winter of 1944-1945, seven nurses and five physicians had acute hepatitis during a period of two months. One of the physicians still has chronic hepatitis resulting from this illness.

TABLE 1
CASES OF INFECTIOUS HEPATITIS BY YEARS

	1934	1941	1942	1943	1944	1945	1946	Totals
Icteric	1	1	2	0	9	3	4	20
Non-icteric	0	0	0	0	7	3	12	22
Totals	1	1	2	0	16	6	16	42

A study of our series of 42 cases shows that 22 lived in Shreveport while the remaining 20 came from 18 different communities in North Louisiana, East Texas and South Arkansas within a radius of 100 miles. Ten of these children were referred by the home physician, while the remainder had previously been under our care. This might indicate that a number of other cases of the disease had been treated by the home physicians in the respective communities, and reports from some of the families indicated that such was the case. In 1946, twelve of the individuals (ten children, two mothers of affected children) lived in the same general area in Shreveport and became ill during the months of May through July. Half of them had possible direct contacts, through school, play or family associations, but all other cases appeared to be sporadic.



The seasonal incidence (fig. 1) shows a spread throughout the year, with greatest prevalence during the summer months.

A survey of age incidence (table 2) shows that almost half (20) of the affected children were between five and ten years of age, 15 were between one and five years, five were between ten and fifteen years and two were adults (mothers). No infants under one year were seen with the disease. Since 30 of these cases occurred in our regular clientele, interpretation of these figures in accordance with our case load distribution by ages is necessary. Recent figures for the Study of Child Health Services show that the pediatric case load is greater in the one to five year group than in the group above six years; hence the true incidence of hepatitis in older children is probably even higher than indicated by our present series.

TABLE 2

CASES OF INFECTIOUS HEPATITIS BY AGE

	1-5 yrs.	6-10 yrs.	11-15 yrs.	Adult
Icteric	6	11	3	0
Non-icteric	9	9	2	2
Totals	15	20	5	2

HOMOLOGOUS SERUM HEPATITIS

The possibility that several of our cases of hepatitis may represent homologous serum hepatitis must be considered. In February and April, 1944, two boys of six and seven years received intravenous scarlet fever convalescent serum, 20 c.c. each from the same lot, for treatment of this disease. After intervals of 93 and 60 days respectively, they became ill with acute hepatitis, icteric in one instance, non-icteric in the other. No other children given the serum developed jaundice and the serum center[9] reports that no other cases of homologous serum hepatitis developed from this lot of serum, so that the hepatitis in these two instances was probably coincidental. One other child in our series developed hepatitis 189 days after receiving a transfusion, but this would be an extremely long incubation period; furthermore, this donor has no history of hepatitis and other transfusions from the same donor were not followed by jaundice. No other children in our series had a history of serum or blood administration within a year prior to inception of the hepatitis.

SYMPTOMS AND CLINICAL COURSE

A: ICTERIC CASES

Twenty children developed clinical icterus; an equal number of the children and the two mothers had non-icteric hepatitis. Those who developed jaundice were usually sicker, the jaundice being preceded by a preicteric stage of fever, ranging from 99 to 104 degrees F. for a period of two to five days, anorexia, malaise and usually pronounced abdominal symptoms, nausea, vomiting and upper abdominal pain being the more frequent. Occasionally constipation loose stools, coated tongue or general abdominal soreness were mentioned and the stools were noticeably light colored in six instances. Other symptoms were headache, restlessness or irritability, epistaxis, dark colored urine, and weight loss. In two instances, chills accompanied the initial fever and malaria was suspected, but no parasites were found and therapeutic trial was without effect.

While fever usually had subsided in from one to several days before jaundice appeared, low grade fever sometimes persisted. In three instances, the children had acute upper respiratory infections beginning ten days to two weeks before inception of the jaundice, with fever for several days, then an afebrile period of a week to ten days during which the respiratory symptoms persisted, together with malaise and increasing abdominal symptoms before icterus appeared.

In four of the children, icterus was noted only in the sclerae, while in the remainder the skin and sclerae were icteric in varying degree. A "sallow" color was often noted by the parents before clinical icterus became apparent. Itching or scratch marks on the skin were present in three instances and two children had evanescent skin rash on the first febrile day. The duration of the jaundice averaged ten days to two weeks, although three weeks was not unusual and two children were slightly jaundiced for a month. Anorexia or lack of vigor sometimes persisted for a week or two after icterus cleared. In other cases, the children were jaundiced for only a week and seemed to recover very rapidly. The duration of the entire illness in the icteric group varied from ten days to two months, with the usual course being two to three weeks.

SYMPTOMS AND CLINICAL COURSE
B. NON-ICTERIC CASES

The clinical symptomatology in the non-icteric group was milder. Fever was often absent or unrecognized, although recordings of 99 to 101 degrees F. for periods of one to three days were not unusual and two children had temperatures of 103 to 104 degrees. Malaise, fatigue, irritability, dizziness, headaches and vague abdominal complaints were the rule, but some of the patients had more severe gastrointestinal symptoms — vomiting, upper abdominal pain, loose stools. Acholic stools were noted in five instances. The milder cases suggested the clinical picture formerly called "biliousness." Although no clinical icterus was in evidence, the skin was noted to be "sallow" or pale in about half of the cases, and a history of itching or scratching was secured several times.

Antecedent illnesses included tonsillitis, upper respiratory infection, measles and— in two instances — acute gastroenteritis (virus type), all occurring within a month before the hepatitis. Two patients referred from other towns had histories of siblings ill several weeks before, one diagnosed as virus pneumonia, the other as acute upper respiratory infection, with clay-colored stools noted during the illness.

PHYSICAL FINDINGS

The findings on examination were often disappointingly few, except for the skin and scleral coloration. Slight adenopathy, especially cervical, was noted rarely. The liver was perceptibly enlarged in 10 of the icteric cases and four of the non-icteric, noted as two to three finger breadths below the costal region and tender in a few instances. Soreness or mild rigidity of the upper abdominal muscles was noted several times. The spleen was palpable in seven instances, extending one to two finger breadths below the costal margin. Slow heart rate, skin rash and scratch marks on the skin were other occasional findings. When vomiting had been excessive. mild dehydration, reddened or cracked lips and acetone odor were present.

LABORATORY FINDINGS

In view of the repeated demonstration [1, 8] that 50 per cent or more of the individuals infected with acute hepatitis in an epidemic never develop visible jaundice and that the symptom complex in the absence of jaundice may simulate a number of other diseases, we were interested in discovering which laboratory procedures are reliable and adaptable for use in our clinic. No single test is diagnostic of this disease, but correlation of the routine blood examination, urine tests and two or three selected liver function tests with the clinical picture will at least give evidence of liver dysfunction strongly presumptive of infectious hepatitis.

Blood counts: complete blood counts were made one or more times in 28 of the 42 patients we have seen. The red blood cell counts and hemoglobin estimations were within the normal range, unless the child had antecedent anemia, and the erythrocytes showed nothing unusual. This finding is in contrast to the pallid appearance of the skin in non-icteric individuals. The leukocyte count varied considerably, even in uncomplicated cases, with a range from 3,200 to 15,000 cells per cu. mm. However, moderate leukopenia was the general rule, with 17 of the 23 uncomplicated cases having counts of 7,000 or below. Distribution of the leukocytes also varied considerably, with neutrophiles varying from 13 to 75 per cent, lymphocytes from 21 to 66 per cent, monocytes 1 to 15 per cent and eosinophiles 1 to 11 per cent.

The striking finding on examination of blood smears was the appearance of the lymphocytes, an appearance first noted in the instance of children with acute (virus) gastroenteritis during the epidemic of 1943-1944. For a time the technicians called us to the laboratory almost daily to discuss the unusual lymphocytes, which we came to speak of as having the "virus look", especially after we noticed the same type of cells in acute hepatitis during the past two years. These lymphocytes appear slightly young, with azure stained granules in the cytoplasm, the nucleus often indented and having clumped chromatin, and vacuoles often appear in the nucleus or cytoplasm. Barker, Capps and Allen[1] mention that 5 to 20 per cent of the lymphocytes in smears from their series of infectious hepatitis were atypical, similar to those of infectious mononucleosis, and one of these authors[10] later informed us that similar lymphocytes were frequent in studies of acute virus gastroenteritis in army personnel. No published report of this observation has come to our attention. Undoubtedly the abnormal lymphocytes seen in these three diseases are similar, although in our experience the changes in acute mononucleosis are more striking, and the lymphocytes seen in hepatitis and gastroenteritis of virus origin do not often have the typical cartwheel arrangement of nuclear chromatin.

Urine examination: The urine is usually dark colored during the period of initial illness, well before jaundice occurs. In the non-icteric, this dark color may last for only a few days, but in sicker patients, it persists for some time. A trace of albumin is not infrequent. The tests of importance, however, are those for bile salts and bilirubin, which have been positive in all of our cases, icteric and non-icteric. We have found that as the child improves, the jaundice clears, then within a few days the urine bilirubin test becomes negative, but a positive bile test is likely to persist several more days. Liver function tests are likely to show activity for quite a period of time after all urine tests are normal, so that positive tests for bile or bilirubin in the urine are of importance in diagnosis but not in judging complete recovery.

The bile tests which we have used are the nitric acid ring test or old nitric acid saturation of filter paper, to which a drop of urine is added. Surveys which we have made and will report in detail later, however, demonstrated that these tests are not as specific and apparently are subject to more variation in interpretation than the bilirubin test. Even with the latter, proper technic seems to be of importance to avoid misinterpretation. We first used Myers'[11] modification of Franke's methylene blue test for bilirubin, in which two drops of Loeffler's methylene blue are added to 10 c. c. of urine. If a green color results, the test is considered positive and is repeated with increasing dilutions of urine until the dilution is reached in which two drops of the reagent produces a blue color, thus giving a roughly quantitative test. In surveys of clinic patients and hepatitis contacts with this test, irregular results and high percentage of positives led to the adoption of a similar test described by Gellis and Stokes[12] which in our laboratory has seemed much more satisfactory. This technic is as follows: to 5 c. c. of urine (preferably prebreakfast specimen) is added two drops of an 0.2 per cent aqueous solution of methylene blue chloride. If a green color results, more drops of the methylene blue are added and the last drop required to convert the green color to blue is recorded. If more than five drops are required, the test is considered positive. Gellis and Stokes found close correlation between results of this test and the icterus index in 77 cases of hepatitis, with positive methylene blue tests preceding clinical icterus by one to six days. Although less sensitive than the Harrison spot test and open to some objection in that the green color is due to a blend of blue and yellow colors and not to a chemical combination between bilirubin and methylene blue,[13] its simplicity makes it valuable for clinical use in any laboratory.

Liver function tests: There are numerous tests of liver function and obviously the clinician will find it neither expedient nor necessary to secure a large number of these. It seems better, therefore, to attempt to evaluate them in the light of information secured and adaptability to clinical use. For example, the icterus index is largely confirmatory and of some value in following a case closely, but not very important in diagnosis, since the degree of clinical jaundice and the icterus indices are closely correlated. In our series, for example, values of 14 to 45 units were found in icteric cases; four to 18 in the non-icteric (table 3). The van den Bergh is of more value, especially in differentiating hepatitis from the icterus of hemolytic blood dyscrasias, as a positive direct reaction usually occurs in the former but not in the latter. A prompt direct reaction was secured in eight to ten patients in which we used this test, including the non-icteric (table 3). Quantitative serum bilirubin was in excess of 2 mgm. in the icteric patients but in six non-icteric patients it was below this figure. Serum protein determinations in two instances showed lowered albumin and elevated globulin values, but these determinations are time consuming in view of limited information secured.

Numerous other tests—bromsulfalein retention, alkaline phosphatase, hippuric acid excretion, glucose tolerance, cephalin cholesterol flocculation test of Hanger, thymol turbidity, serum cholesterol, serum amylase and prothrombin time, to mention only a few—have been used by investigators. All liver functions may be disturbed in varying degree during acute hepatitis, even in the mildest, non-icteric form. We have used the flocculation test of Hanger, as reports had indicated its adaptability to clinical use and comparative reliability as an indicator of liver damage. All of our icteric cases which were tested and eight of the 12 non-icteric cases showed 2 to 4 plus Hanger readings (table 3). In the large series of cases reported by the Hepatitis Commission[1] Hanger tests were positive in 50 to 75 per cent during the prodromal stage, in 90 per cent during the acute icteric stage, and in 53 per cent of the non-icteric cases. In their series, the bromsulfalein dye retention proved to be the most reliable test, while others consider the thymol turbidity test to be more sensitive.[14]

TABLE 3
LABORATORY FINDINGS IN 12 CASES OF NON-ICTERIC HEPATITIS

Patient	Age in years	Leukocyte count Cells per cu. mm.	Percentage of lymphocytes	Icterus index	Van den Bergh qualitative	Serum bilirubin (mgm. per 100 cc.)	Hanger's cephalin cholesterol flocculation	Urine bile	Urine bilirubin
M. M.	11	3,950	59				2+	Pos.	Pos.
K. M.*	42			5.8	Pos.		3+	Pos.	Pos.
E. W.	10	4,100	54**	5.0	Pos.	1.4	4+	Pos.	Pos.
D. W.*	41			8.0	Pos.		Neg.	Pos.	Pos.
J. M.	10	21,000†	35**	8.5	Pos.	1.8	Neg.	Pos.	Pos.
M. A. M.	11	5,000	52	5.0			2+	Pos.	Pos.
R. L.	4	6,000	66**		Trace		2+	Pos.	Pos.
C. E. A.	6	3,200	63**		Trace	0.26	3+	Pos.	Pos.
B. S.	6				Pos.	0.76	Neg.	Pos.	Pos.
J. S.***	4			4.7	Neg.	0.23	Neg.	Pos.	Pos.
J. B.	3				Pos.	0.32	3+	Pos.	Pos.
J. G.	5	8,900	18	18.8	Pos.			Pos.	Pos.

* Mothers of M. M. and E. W. respectively.

** Atypical lymphocytes seen on blood smears.

***Sister of B. S.

†Patient also had acute maxillary sinusitis.

PROPHYLAXIS AND TREATMENT

Since this disease is demonstrably contagious, care should be taken in disposal of excreta from the individual case and children should be kept away from the infected individual. In the presence of epidemics or in endemic foci, food and water supplies should be carefully checked. Individuals with histories of jaundice should not be used as donors for transfusions or sera.

Stokes and Neefe[15] and Gellis et al.[16] have demonstrated, in the presence of epidemics of infectious hepatitis, that the administration of gamma globulin to contacts reduced the incidence of the disease by 60 to 80 per cent, and modified the illness in the remainder. The dosage of gamma globulin used was 0.15 c. c. per pound body weight in children, 10 c. c. in adults, in a single injection.

In treatment of the active case, two considerations of prime importance are rest and diet. Experiences in the armed forces[1] showed that early and complete bed rest for an adequate period of time decreased the severity and reduced the incidence of delayed convalescence. Bed rest should continue until symptoms have cleared, the liver has returned to normal size and is not tender, the urine is clear of bilirubin and one or more of the liver function tests (Hanger, bromsulfalein, van den Bergh, thymol turbidity) are normal. In case of recurrence of symptoms, as happened in three of our cases, immediate resumption of bed rest is essential.

The diet should be high in protein and carbohydrate, low in fat. The experimental work of Miller, Whipple, Györky and others[17, 18, 19, 20] indicates that a high protein diet is far more protective to the liver than a high carbohydrate diet, in fact, that the latter is probably of value chiefly because of its protein sparing effect. Further study indicates that the sulfur-containing amino acids, methionine especially, or cystine plus chlorine, are the particular substances in the high protein diet which protect the liver. Foods reported to be rich in methionine are casein, serum albumin and egg white.

Eddy[21, 22] found methionine to be valuable in treatment of toxic hepatitis of chemical origin and due to his efforts methionine has been available locally. We have used it in several cases of infectious hepatitis, in dosage of two to three grams daily, apparently with good results. Prior to that time we had advised high proteins, especially skimmed milk (casein), with choline.

Due to fat restriction, extra supplies of fat soluble vitamins are desirable, also vitamin B complex in moderate amounts. Ample fluids should be given in the acute stage and, in the severely ill, intravenous fluids may be essential.

SUMMARY

1. The high incidence of acute infectious hepatitis in the armed forces has been paralleled by increased incidence in the civilian population in this area during the past three years.
2. Non-icteric cases of this disease appear to equal or exceed the icteric. They exhibit the same general symptom complex in milder form and if suspected may readily be demonstrated by proper diagnostic procedures.
3. Tests which are readily available to any good clinical laboratory are adequate for diagnosis of acute hepatitis. Those which we have found to be of especial value are: Leukocyte count with differential study, urine tests for bile and bilirubin (especially methylene blue test for bilirubin), qualitative van den Bergh, quantitative serum bilirubin and Hanger's cephalin cholesterol flocculation.
4. Abnormal lymphocytes found in this disease are apparently identical with those found in acute (virus?) gastroenteritis and very similar to those found in infectious mononucleosis.
5. Important principles of treatment include adequate and prolonged rest and high protein, low fat diet. The amino acid methionine is the important liver-protecting constituent of the dietary protein.

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ACUTE DIARRHEA IN CHILDREN

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Diarrheal disorders are widespread and constitute in the summer months one of the major causes of illness and infant mortality. This is particularly true in the Southern tier of states. The child brought to the physician with a chief complaint of diarrhea may be suffering from any one of a large group of diseases. The recognition of the etiologic agent and the treatment of the acute illness are the two important problems confronting the medical practitioner.

The accurate diagnosis of these diarrheal disorders is a problem for the laboratory, since it has been shown repeatedly that clinical findings in an individual case can do little more than suggest the etiological agent. There is one point, however, which is of some practical value to the physician in the South. Usually a diarrhea which is sufficiently severe to cause the mother to consult a physician is caused by one of two large groups of intestinal pathogens, the *Shigella* and *Salmonella* groups.[1] In all parts of the country that have been studied, the *Shigella* group predominates, but the relative importance of the two varies considerably.[2] In some areas the *Salmonella* infections have constituted less than one per cent of the endemically occurring cases, whereas in others, specifically in the New Orleans area, the *Salmonella* group has been isolated from as high as 25 per cent of the cases admitted to a hospital service. For practical purposes, in the absence of laboratory facilities, the physician can safely assume that something over three-fourths of his cases of acute diarrhea are due to an organism belonging to one of the above groups, and it is therefore good practice to assume that all of these disorders are infectious until proved otherwise. This is particularly important when there are other young children in the family.

DIAGNOSIS

As stated previously, the problem of diagnosis is basically a laboratory problem, and in the last few years this has been tremendously simplified. The smallest laboratories can now do satisfactory enteric diagnostic work if they will avail themselves of the newer tools. Culture media are available in dehydrated form which permit preparation of fresh media on a few minutes' notice. They are specifically designed

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to promote the growth of enteric pathogens, and at the same time inhibit those nonpathogenic organisms which ordinarily are present in large numbers in any fecal specimen. The best medium for the purpose is called SS (*Shigella-Salmonella*) agar. For accurate diagnostic work, it is essential that this medium or a similar one be used.[3]

The selectivity of these media has greatly simplified the obtaining of necessary diagnostic specimens. When using older types of media, it was necessary to have someone obtain a specimen of the freshly passed stool, place it in a container and send it, often with considerable delay, to a selected laboratory. A considerable change in fecal flora usually took place under these circumstances before the laboratory had any opportunity for examining the specimen. By using the new media, it is now possible to obtain a specimen in much the same way as doctors have used for many years in the diagnosis of diphtheria. The media can be used in the office, or in the home, the specimen being obtained at the time of consultation. An ordinary cotton tipped applicator is inserted into the rectum of the patient. This is easily accomplished if care is taken to spread the buttocks as widely as possible before inserting the swab. A sufficient amount of feces will adhere to the cotton, and then the swab is used to plate the material directly on the culture medium. The entire surface of the plate should be covered, using a painting motion. This method has been used successfully in both hospital and private practice for the past four years. In one hospital where the routine practice of culturing each case on admission was in effect, the shortening of time required for a laboratory report was spectacular. It was found that when the rectal swab was taken and plated on admission of the patient, it was possible for the laboratory to give a preliminary report, on the average, 36 hours later. When the attending physician simply ordered a stool specimen to be sent to the laboratory, the average interval between admission and reporting was seven and a half days. When one considers the usual short duration of most of these infections, it is little wonder that physicians have taken very slight interest in enteric cultural work. The patient was either dead or recovered before the doctor had any report as to the results of the culture. By using the simplified method, however, a report can usually be obtained during the acute phase of the illness and at a time when it can probably influence management of the case. This will be brought out in more detail when specific therapy is discussed.

The whole field of enteric bacteriology is a complicated one, and the physician in practice cannot be expected to acquaint himself with all newer developments as they occur except in so far as they have practical application to the treatment of his cases. For this reason, we are not discussing the many different types of organisms which may be encountered in the course of a summer's work. For example, in one area in four months' time we isolated some eight different types of *Shigella* and 22 different types of *Salmonella*. This information is important to the epidemiologist in his search for sources and modes of spread of these infections. At our present stage of knowledge, however, there is little or no practical value to the physician in identification of these organisms beyond the particular genus to which they belong. The genus of the pathogen present is of significance because: (1) the *Genus Shigella* is susceptible to sulfonamide therapy whereas the *Genus Salmonella* is not; (2) the *Shigella* group is for practical purposes a human pathogen and lower animals are not hosts to this parasite. On the other hand, the *Salmonella* group are widespread in the lower animals. This has a considerable influence on any investigation designed to determine the source of an infection.

In summary, the laboratory tests outlined above, namely isolation and identification of the etiologic agent, are the only ones of practical diagnostic value. Agglutination tests have been used, but they, at best, could only give a retrospective diagnosis, and recent studies have indicated that in the individual case it is impossible reliably to interpret positive or negative findings.[4] Complement fixation and other similar tests have not been developed.

When a satisfactory laboratory test has been performed with negative results, other possible causes of the diarrhea should be considered. Usually, however, in the acute illness found negative on culture, the duration is short and the patient's recovery is prompt, with little or no assistance on the part of the physician. The cause in these cases may be extremely variable, but the treatment is essentially the same for all.

TREATMENT

The major problem in the management of diarrheal diseases of children is treatment of the severe dehydration which frequently occurs in these patients, especially in infancy. In addition, acidosis is almost invariably present in these dehydrated infants and must be treated specifically. This problem is greatly simplified if laboratory facilities are available. The critically ill patient should have the CO_2 combining power of the blood determined immediately. In severe acidosis this figure is below 30 volumes per cent and one of the following solutions is indicated to correct the acidosis. Roughly, 1 c. c. per pound of body weight of either one sixth molar sodium lactate, or 1.5 per cent sodium bicarbonate is needed to raise the CO_2 combining power one volume per cent. Thus by subtracting the determined level from the normal of approximately 55 volumes per cent, the necessary number of c. c.'s per pound is obtained for either solution. At least half of the required amount should be given immediately by intravenous injection, and the remainder may be given subcutaneously or intraperitoneally. Dehydration is further combatted by the use of Ringer's solution and 6 per cent dextrose solution in approximately equal quantities. These solutions may be given intravenously or subcutaneously, and higher concentrations of dextrose are possible by vein if given slowly. The use of dextrose solution avoids an over-supply of sodium chloride, and in addition, it is particularly valuable in treating the ketosis and starvation which may be present. A total of 75 c. c. to 150 c. c. of fluid per pound of body weight are needed in the first day in severe dehydration and acidosis.[5]

The intravenous drip method of administration is particularly valuable in these cases of severe fluid loss. In the early stages of administration gravity flow through a 22 to 24 gauge needle may be permitted and after approximately half of the needed volume has been administered the rate of flow is cut to approximately 5 to 10 drops per minute. Where the drip method is not possible, single intravenous injections of 10 to 30 c. c. per pound may be given over a 20 to 30 minute period. This dose may be repeated every four hours until the dehydration is overcome. A large volume can be given subcutaneously even in small babies when the dehydration is marked. This method is frequently the most convenient for the practitioner who has to handle these patients in the home. Also where vomiting is not marked, much of the necessary fluids can usually be given by mouth if care and patience are exercised.

When the laboratory facilities for CO_2 determination are not available, some arbitrary rule of thumb must be used. It has been found that severe acidosis can usually be overcome by 15 c. c. per pound of one-sixth molar sodium lactate solution or 1½ per cent sodium bicarbonate solution given intravenously, followed by an equal amount administered subcutaneously. Ringer's and dextrose solutions are then given in the same manner and quantity described above.

When the fluid and electrolyte balance has been reestablished, about 60 c. c. of fluid per pound are necessary daily to maintain hydration in the normal infant. With continuing diarrhea, more than this is required, as well as approximately 15 c. c. per pound of one-sixth molar sodium lactate, to prevent the recurrence of acidosis. Ketosis may be avoided by 0.5 gm. of dextrose per pound of body weight each day.

The cases of diarrhea which fail to do well under the above regime may be suffering from a lack of potassium. This has been brought out by the investigations of Darrow and his co-workers,[6] and a new solution designed to take care of this deficiency is now being investigated.[7] It promises to be of great value and probably will be standardized in the near future. At present, however, it is not generaly available.

When dehydration and acidosis are moderate, lactate-Ringer's is the solution of choice. This may be given orally except in cases of vomiting and in infants when parenteral injection is necessary because of the rapidity with which a dangerous condition may develop. It is much easier to *prevent* severe acidosis than to treat it once it has arisen. The mild cases are treated satisfactorily by forcing fluids, water or normal saline, by mouth.

In cases of diarrhea with excessive vomiting, there is considerable loss of chloride ions from the body. This lack is usually adequately made up by chlorides in the solutions given for dehydration. Ringer's solution is especially good and may even be tried by mouth in small quantities, one teaspoonful every fifteen minutes, and gradually increased as tolerated.

After body fluids have been restored, a whole blood transfusion, concentrated plasma, or amino acid hydrolysates may be extremely valuable. This is particularly true in the poorly nourished patient whose serium protein is very low. This treatment should be repeated as necessary, whenever recovery is slow; and in such cases, parenteral vitamin administration also is helpful.

Various sulfonamide preparations have been found to be effective in the treatment of *Shigella* infections. Since a high percentage of severe cases are caused by a member of this group of organisms, the use of a sulfonamide preparation is often indicated at once, even before a definite diagnosis is finally established.

Absorbed sulfonamides have been shown to be most effective, and of these, sulfadiazine, sulfapyrazine and sulfathiazole have been shown to be effective in the order named.[2] The dose should be approximately ¾ to 1 grain per pound of body weight daily. It is given in divided doses four to six times per day. Usually to insure more rapid therapeutic levels of the drug, the initial dose is doubled. When stool cultures are obtained, this therapy should be modified to conform with the findings. For the *Shigella* group of organisms, the sulfonamide should be continued until two negative cultures have been obtained, or for seven days, whichever comes first. Occasional organisms which have not responded by the end of seven days to such therapy are usually resistant strains, and continued administration of the drug is not beneficial. Since the *Salmonella* group do not respond to these drugs, isolation of this organism is an indication to stop sulfonamide therapy. A negative cultural result may be due to technical difficulties and in such a case the physician should be guided by the clinical results obtained up to that time. If the patient is unable to take medication by mouth, 0.5 per cent sodium sulfadiazine may be given parenterally along with other fluids. Dose schedules remain the same. Therapeutic levels of sulfadiazine are present in the stools within a matter of hours after parenteral administration. When using these absorbed sulfonamides, particularly sulfadiazine, it is important that the acidosis be vigorously combatted, since an alkaline urine is advisable for prevention of urinary complications.

Sulfasuxidine is the best of the poorly absorbed sulfonamides now available. It is effective against the *Shigella* group, and it has the advantage of an extremely low rate of complications. This drug to be effective must be used in large doses approximately five times those given above for the absorbed sulfonamides.

General measures used in treatment of any diarrhea include rest, a fasting period of 12 to 24 hours, and careful attention to the diet. Sedation is frequently useful when pain or vomiting is marked, or the patient is particularly restless. Cathartics should not be permitted.

The diet in general should be low in fat and roughage, high in protein. Continued breast feeding is satisfactory when possible. The artificially fed babies should be given a dilute formula of skimmed or evaporated milk. The addition of protein hydrolysates or the use of one of the protein milks may be valuable in the early stages of treatment. The formula in these children may be made up with lactate-Ringer's solution as an aid in combatting acidosis. It is necessary when beginning feeding after the period of starvation to keep the feedings small and frequent, gradually increasing as the patient improves until the normal formula is resumed. The same is true with other children.

A number of special preparations, such as apple powders, foods combined with agar and kaolin mixtures have been used with varying degrees of success. They all seem to be helpful in the control of diarrhea.

SUMMARY

The management of the severe case of diarrhea requires a good laboratory for best results. The laboratory provides the means of intelligently combatting acidosis and dehydration, the usual cause of death when it occurs. Second, it provides a basis for intelligent use of specific therapeutic agents now available. With or without such facilities, however, the important thing to remember is that energetic treatment of the dehydration and acidosis does more to save life and shorten illness in young children than any other single measure. This combined with specific sulfonamide therapy will effectively bring about recovery of all but the most severe cases.

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CHRONIC COUGH IN CHILDREN*

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Like so many common symptoms, cough very often fails to fit into pathognomonic patterns, respond to recommended remedies, or receive the respect it deserves. Regardless of the cause, a persistent or severe cough can itself lead to destructive changes in respiratory surfaces, bronchial dilatation, and emphysema, as well as a number of other more remote but equally dangerous effects. With cough as a chief complaint, the list of possible diagnoses is long, and little point would be served by including it here; the more careful the clinical study, the longer the list becomes. Perhaps no common symptom is passed over lightly so often or treated so frequently without establishing a satisfactory etiology. It is as though physicians justifiably tire of continuously facing this diagnostic challenge and so take refuge in merely treating the symptom. Cough is essentially a protective mechanism. A physician many times does harm by injudiciously prescribing specific medication to suppress rather than aid the reflex.

Students, like their textbooks, place tuberculosis and bronchiectasis high on their differential diagnostic lists; commendable as the practice may be, we can certainly all agree that these are relatively rare causes of cough among children of the age when the complaint is commonest. Fortunately, these two very serious causes can be excluded readily by dependable objective means, but then there is a slump in diagnostic enthusiasm—what next? Forget about it and suppress the reflex? Tell anxious parents they need not worry? Depend on nature, time or a change in the weather?

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Only very occasionally can the answers to these questions be "yes"; in most cases serious enough to be brought to medical attention, our work has only begun.

The pattern in the act of coughing is familiar: preliminary inspiration, closure of the glottis, increasing intrathoracic pressure, sudden opening of the glottis, and a strong expulsive effort. Whereas it certainly may be voluntary and though it may sometimes be withheld, it usually appears to be entirely reflex in nature. The younger the child, the more *inefficient* a cough appears to be. Expulsive efforts often seem purposeless, poorly directed, and ill-timed; tiring paroxysms are apt to follow apparently minor stimuli; major stimuli may be followed by only feeble, inefficient hacks. Aspiration of anything that happens to be present in the mouth, nose, or throat commonly occurs with the next inspiration. Stimulating impulses may arise from intrinsic or extrinsic irritation of the respiratory mucosal surfaces, excitation of nerve endings in the pleura, vagal afferents in other areas such as the esophagus, stomach, or external auditory canal (Arnold's nerve). Pressure adjacent to these structures may also produce cough. There are about seven other means by which irritants and secretions may be removed from the respiratory tract; the first three of these—ciliary action, bronchiolar peristalsis, and phagocytosis in the alveoli—might be termed "insensible" and apparently act adequately alone to remove normal secretions or small aspirated particles; cough probably occurs only when these three physiologic processes are overtaxed. The other four—postural drainage, hyperventilation, suction technics, and physical or chemical measures designed to reduce the viscosity of secretions or exudates—are ordinarily employed in turn only to aid the overtaxed cough.

Productive or not, cough of severe recurrent or paroxysmal nature in young patients often appears to require an inordinate amount of physical work causing all too apparent exhaustion. Obviously inadequate pulmonary ventilation, cyanosis, or convulsions are not unusual and may be accompanied by serious degrees of anoxia, respiratory acidosis, and petechial or gross hemorrhages in the brain or meninges; extensive encephalopathy has been ascribed to resultant circulatory changes.

In case histories, one is struck with the large number of potential inciting factors that may be elicited; it is usually difficult to ascribe chronic cough in a child to any single one. In many cases, familial allergic features (real or imagined) justify suspicion of atopy as a major or contributing cause. Tuberculophobia, which so often brings many of the milder cases to medical attention, is usually based on previous unfortunate familial occurrence; parents who have had experience with other serious or chronic pulmonary disease may be similarly apprehensive. A cough which is continuous, increasingly severe and productive, and aggravated by change in position, is particularly ominous; beginning in infancy and accompanied by recurrent diarrhea and wasting, there is legitimate reason to suspect fibrocystic disease with its familiar sequential pattern. A cough which follows aspiration of a foreign body, any operation on the upper respiratory tract, anesthesia, or inhalation of common irritants such as oils, kerosene, or powders, must always be regarded seriously.

Histories are usually replete with distinct evidences of chronic or recurrent infections and obstruction in the upper respiratory tract; these precede or accompany exacerbations of cough and fever of variable degree and duration. These episodes are often justifiably diagnosed as bronchitis or pneumonia, initiated by fresh "colds" or any other common infection. There are many corroborative complaints attesting to the chronicity or frequency of these: poor appetite and weight gain, nervousness, fatiguability, and behavior problems of seemingly endless variety.

From careful description of a cough, the clinician often gains diagnostic information of localizing value, even though inaccurate accounts by distraught parents may require meticulous interpretation and evaluation. There is no doubt that a careful history — neglected as it has been in these past few busy years—will almost always furnish more pertinent clues to correct diagnosis than any other portion of the clinical study.

Though physical examination may yield surprisingly negative results, it more often discloses many abnormalities related as cause or effect to the major complaint. Signs of chronic infection are common, though variable in degree: faulty posture, small stature, anemia, poor muscle tone, weight loss, or inadequate gain. There is usually unmistakable evidence of disease about the pharynx, nasopharynx, nose, paranasal sinuses, ears, or regional cervical nodes. Visible, audible, and palpable obstruction to the nasal airway may exist. In many of these patients, characteristic pale, boggy, glistening nasal mucosae lend support to suspicions aroused by an allergic history. Such evidence, even when accompanied by distinct infection, is best documented by careful nasal smears prepared with Giemsa's or Wright's stain; nasal eosinophilia can be demonstrated frequently in these cases but is rare in nonallergic individuals. By simple testing, obstructive deafness can often be demonstrated; here otoscopic examination shows some retraction of tympanic membranes or signs of middle ear infection.

Conventional examination of the pulmonary fields is usually disappointing. Although respiration may be slightly increased, percussion note a bit hyperresonant, and breath sounds somewhat harsh, wide physiologic variations make these difficult to appraise. Scattered rales may be heard about the pulmonary hilar areas or bases but usually clear by the time of day these children are brought to the doctor's office; during exacerbations demanding home visits, indications of pulmonary disease are usually clearcut and multiple.

Whereas roentgenographic examination is a most valuable adjunct in studying these cases, it is often improperly employed as a shortcut to supplant equally dependable but more time-consuming diagnostic measures. Interpretations should always be correlated with other clinical findings, rarely trusted alone. To reenforce careful examinations, views of the chest and paranasal sinuses are taken almost routinely in any complete study of chronic cough in a child; lateral views of the nasopharynx are also occasionally useful. "Surprises" should always lead us back to the patient to recheck our observations and technics of physical examination but never to depend abjectly on the x-ray.

Appropriate serial tuberculin tests should be performed in all children, regardless of whether or not cough is present. Positive reactors may be disclosed more frequently among children with chronic cough; then the real problem is to differentiate between active or quiescent, infectious or noninfectious, and primary or reinfection forms of tuberculosis. Any serious infection responsible for chronic cough will generally be accompanied by measurable evidences such as fever, increased sedimentation rate, leukocytosis, and polynucleosis. If the cough is productive, proper examination of the sputum may directly and correctly define its cause. From infants and young children sputum is best obtained by nasopharyngeal swab or esophageal aspiration. In addition to the usual obvious indications for such study, occasional examples of mycotic infection can be discovered by this means.

Whenever there is reasonable evidence of pulmonary suppuration, complete bronchographic study after instillation of lipiodol should follow as a matter of routine; only by this means can the presence of bronchiectasis be established with certainty.

When chronic cough is accompanied by symptoms and signs suggestive of the celiac syndrome, fibrocystic disease must be considered and pancreatic enzymes in duodenal contents investigated. Indeed, in some examples of this condition, symptoms of intestinal disorder are overshadowed by serious pulmonary manifestations; the presence of inspissated secretions accompanied by dilatation, atrophy and fibrosis of glandular structures all over the body furthermore suggests that such changes in pancreatic acini are only incidental to a generalized disease not yet completely understood. Examination of the feces should be a routine part of any investigation to determine the cause of chronic cough; common parasitic diseases (ascariasis, strongyloidiasis, and hookworm infection), in which the responsible organism ruptures the pulmonary capillaries and alveoli, may be recognized. These undoubtedly produce specific respiratory symptomatology as well as constitutional effects.

After analysis and correlation of thorough diagnostic procedures, specific therapy may be selected. While such studies are in progress, however, diligent efforts should always be made to treat contributory or associated infections, relieve obstructions, aid in removal of secretions, and improve nutritional deficits. The simplest technics are still most generally effective: intelligent administration of nose drops, postural drainage, hyperventilating exercises, and increased humidity.

In children, use of any of the common agents for shrinking nasal mucosae should be interrupted for about twelve hours every few days to minimize undesirable secondary irritative effects. The dropper is preferable to the spray, and accumulated secretions should of course be removed before drops are instilled. The simplest and generally most efficient technic (Parkinson) employs the lateral head-low posture. There is little or no justification for including various sulfonamides in nose drops or sprays. Aerosol solutions of penicillin or streptomycin are certainly beneficial in cooperative selected patients. Most infants and young children prefer to sleep in the prone or even knee-chest position, so effective for clearing secretions from the large respiratory passages; even better drainage can often be secured by elevating the foot of the bed a few degrees. With a little ingenuity, simple hyperventilating exercises attractive to the child can usually be devised, ranging all the way from balloon-blowing to strenuous trapeze exercises.

Increased humidity—obtained by a number of modifications of the old-fashioned familiar croup tent — is helpful often enough to deserve trial along with these other simple procedures. Care should be taken to avoid drafts or undue exposure when steam is being used.

The common "tonics" ordinarily employed are rarely effective in the presence of chronic infections. The rather severe hypochromic anemias seen so frequently can be blamed about equally on infection and deficiency and are best treated directly by transfusion. Various hematinics and vitamin mixtures have a valuable place after such preliminary correction, and their use is certainly to be encouraged in the usual prolonged convalescence.

Unless specific local indications exist, adenoidectomy or tonsillectomy has little to offer the child with chronic cough. Certainly when there are allergic manifestations, this operation should always be delayed until the effect of environmental changes, removal of offending substances, or desensitization has been observed.

With or without demonstratoin of specific sensitivities, the child whose chronic cough appears in any way related to nocturnal environmental influences deserves the opportunity of sleeping in a room from which all possible offenders have been removed. When correlation of positive skin tests and symptoms can be demonstrated, elimination of allergens is generally preferable to desensitization in the young child; unfortunately, the former may be difficult or impossible. The effect of newer antihistamine preparations has not been critically analyzed in respiratory allergic disorders of children, but is certainly promising enough to deserve trial.

When a cough is severe and obviously not accomplishing any desirable function, when its cause has been removed but it persists as an irritative or "habit" mechanism, or when it disturbs necessary periods of rest, adequate sedation may be advisable. Phenobarbital is still the most useful sedative in pediatric practice. Belladonna or opiates should rarely be used, as they increase the viscosity of secretions; when sputum is scanty or absent, however, small doses of codeine may be indicated.

An adequate fluid intake, combined with increased humidity, liquefies sputum more efficiently than the best expectorant drugs: ammonium chloride, ipecac, and potassium iodide. These are all difficult to disguise, and are frequently refused by young patients who appear to need them most. Every physician has his own favorite prescriptions; I know of none that is always successful.

No attempt has been made in this paper to review all the causes, diagnostic studies, and therapeutic measures for chronic cough in children. Such a work would fill several volumes. This common pediatric problem illustrates beautifully the need for seeking and removing the cause while treating the symptom. Perhaps in academic circles we seek too much and treat too little, while in practice the converse criticism often applies.

DISCUSSION

Question: What is the treatment of cystic disease of the lung, especially a large cyst of one lung.

Answer: Such cases are unusual. Treatment consists essentially of surgical removal at the earliest possible date. Once the inevitable infection has occurred, therapy is more difficult and hazardous.

Question: Remark on the use of CO_2 to liquefy secretions and aid cough.

Answer: I suppose you mean 5 per cent CO_2 in oxygen; this may be difficult to use adequately in small children, and I have no conviction that it is more valuable in the therapy of cough than the common alkaline cough mixtures.

Question: In chronic cough of undetermined cause, do you think that the bacterial vaccines on the market are of some benefit?

Answer: Certainly the literature is confusing on that point. From personal experience, I have yet to see such a case I believe has been benefitted by the use of commercial bacterial vaccine. Then too, it is difficult to divorce the effect of such vaccines from any other therapy the physician has employed. From an immunologic point of view, we have little reason to expect results from vaccine.

You are perhaps all familiar with the work of Diehl and his associates—for a large group of University students, the use of oral or injectable "cold vaccines" accomplished nothing in a well controlled evaluation of these products.

MENINGOCOCCAL ADENITIS

CASE REPORT WITH DISCUSSION

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Chronic localized complications of meningococcal infection outside of the nervous system are not uncommon. Arthritis[12] has been encountered perhaps most frequently, with reports giving figures varying from 4.6 and 20 per cent as the proportion following meningococcal infections of all types. Endocarditis,[9] conjunctivitis,[11] panophthalmitis,[5] urethritis,[4] epididymitis,[1, 8] salpingitis,[8] parotitis,[5] purulent pericarditis[5] and nephritis[1] have all been reported as complications of meningococcemia. From all of the instances cited, Neisseria intracellularis has been successfully isolated.

The case here described is, to our knowledge, the first report of suppurative adenitis due to the meningococcus and presumably secondary to chronic meningococcemia.

CASE REPORT

An eleven year old white school girl, E. H., was admitted to Charity Hospital November 6, 1945. The chief complaint was fever, which had persisted for two weeks.

Onset and Course: On October 24, 1945, two weeks before admission, the patient returned from

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school with pain in the right popliteal space. That night she had fever of 102° (orally) but the pain disappeared. Fever continued during the next two weeks, however, and on November 5 pain developed in her right elbow. Her temperature rose to 105°, and she vomited several times. All pain had disappeared by the next morning but the parents brought the child to the hospital. There had been no swelling of the joints nor any other abnormal manifestations.

The family consisted of the father, mother and seven siblings all of whom were well. The child's past history was non-contributory.

Physical Examination on Admission: Temperature 101.4°, pulse 105, respirations 28, weight 60½ pounds. Examination revealed a well nourished, well developed white girl, slightly pale, but not acutely ill. Pertinent findings were: (1) Pharynx and tonsils moderately injected; (2) small, firm, discrete, non-tender nodes present in the cervical, inguinal and axillary regions; (3) a soft blowing systolic murmur heard at the apex, not transmitted.

Laboratory Data on Admission: Hemoglobin 12 grams, red blood cells 4,100,000, white blood cells 12,850, polys 78%, lymphocytes 18%, eosinophiles 3%, monocytes 1%. Sedimentation rate 41 mm/hr., corrected (Wintrobe). Urinalysis negative, serology negative, Mantoux (1-10,000) negative at 24 and 48 hours. Blood culture negative, throat culture negative, stool culture negative. Blood agglutinations: Twice negative for *E. typhi*, *paratyphi B.*, *B. abortus* and Proteus X19.

Clinical Course: The impression on admission was "possible rheumatic fever" and treatment was symptomatic.

Throughout her hospitalization the patient had no complaints and did not appear acutely ill. The fever spiked to 105° the first two days and to 103° on the third day. It then hovered around 100° until the eighth day when it reached 103°. On the ninth the highest point was 104° and on the tenth and eleventh, 102°. Thereafter there was gradual decline to normal by the end of the third week, continuing so until discharge ten days later.

The systolic murmur became progressively louder but did not change in character. On November 18, the thirteenth hospital day, the spleen was palpated 1 cm. below the costal margin. It was firm and not tender. On November 26, the twenty-first hospital day, an erythematous, macular rash appeared on the abdomen, chest and back. This lasted for one day and was asymptomatic.

Laboratory Work During Hospitalization: Electrocardiograms taken on November 7, and November 16 were within normal limits. Repeated blood studies revealed no change except for a slowly decreasing sedimentation rate to 7 mm/hour on December 1. Repeated urinalyses showed no abnormalities. Blood cultures taken on November 9 and on November 16 remained negative. Brucellergen intradermally yielded no reaction. Urine culture and repeated stool cultures showed no pathogenic organisms.

The patient was discharged on December 5, 1945, after four and a half weeks in the hospital without further clue as to diagnosis.

Interval at Home: She was seen in the outpatient department on December 20, 1945, two weeks after discharge, at which time her temperature was 100°. She was said to have been having low fever every afternoon for one week. She had also had a macular rash on trunk and thighs for the three days before re-admission. This rash apparently was similar to that which she had had on the ward. The patient was sent home for Christmas and was next seen on December 27, when she was re-admitted to the hospital.

Second Admission: Afternoon elevation of temperature had continued during the week before re-admission and six days before admission a slightly painful lump had appeared in the left groin. On examination the temperature was 99.4° and a soft apical systolic murmur was present. There was a slightly tender mass, two inches by four inches, in the left inguinal region, which was not fluctuant, red or hot. It was thought to be a lymph node. No vaginal pathology or leg lesion was present and no other glands had changed.

The blood picture was essentially the same except that the sedimentation rate was now 14 mm/hour, corrected. The urine, Mantoux (1-100), Frei test, and electrocardiogram were negative.

Course in Hospital: During the next two weeks the temperature was not elevated but the mass persisted. The lymph node was thought to be softening and on January 7, 1946, ten days after admission, sulfadiazine, 1 gm. t.i.d., was started. The gland was fluctuant on January 10, 1946, three days later, and was aspirated, yielding greenish pus. Small bodies suggestive of gram negative diplococci were observed in direct smears of this material.

While waiting for growth of cultures which had been made from the aspirated pus, sulfadiazine was continued at the same dosage. On January 13, however, three days after aspiration and six days after starting sulfadiazine therapy, the temperature went up for the first time to 102°. The next day it reached 104.4°. On the following day the abscess was incised and drained, yielding thick green pus. In contrast to the first specimen, no organisms were seen on direct smear. The dosage of sulfadiazine was increased to 1 gm. every four hours, but during the next two days the temperature again reached 104.4° and a fine macular erythematous rash appeared. A blood count showed 4,000 white blood cells per cu. mm. and it was thought the patient was having a drug sensitivity reaction. Sulfadiazine was therefore discontinued and penicillin 20,000 units was given every three hours for six days. The temperature gradually returned to normal after four days of this therapy. The wound healed steadily and was closed in about two weeks.

After three weeks of normal temperature it was decided to test the validity of the assumption that the febrile episode was due to sulfadiazine sensitivity. A single dose of 1 gm. of sulfadiazine with 2 gms. of sodium bicarbonate was followed within two hours by nausea, vomiting, malaise and a fever of 102.8° which returned to normal over the next 36 hours. No rash was noted.

No further untoward event took place and the child was discharged on February 15, 1946, after a hospital stay of seven weeks.

She has been followed in the out-patient department since discharge and has continued in good health with no recurrence of symptoms.

The final diagnosis was: (1) Suppurative lymphadenitis, meningococcal; (2) chronic meningococcemia; (3) sulfadiazine sensitivity.

Bacteriology: Because of the observation of gram negative diplococci in the direct smear of aspirated pus, special cultural procedures were undertaken. The aspirated material itself was incubated and the usual media used in this laboratory, infusion broth, infusion agar and infusion agar with 1 per cent human blood, were inoculated. In addition, certain special procedures were followed in an effort to isolate anaerobic bacteria. Brain-heart infusion broth and Brewer's Thioglycollate Broth (Difco) were inoculated and kept in the standard incubator. These media have, on many occasions, grown anaerobic or partially anaerobic bacteria successfully. Finally, chocolate agar plates were seeded and placed under increased CO_2 tension.

It was only from the chocolate agar—increased CO_2 procedure that growth was obtained. From material aspirated on January 10, 1946 and the pus from incision and drainage on January 15, very tiny, smooth translucent colonies were obtained. Organisms from these colonies were typical gram negative biscuit shaped diplococci. The oxidase reaction was positive. Fermentation reactions showed acid production with both maltose and glucose.

A culture was sent to the National Institute of Health for further identification and agglutinations with type specific sera and was reported as meningococcus, Type I.

DISCUSSION

In view of the absence of any positive bacteriologic data on the patient's first admission one can only speculate on the course of events. With no findings referable to the central nervous system and no history of a rash, meningococcemia was not suspected. The rash which was reported on the twenty-first hospital day of the first admission occurred after the temperature was normal, was only transitory, and was certainly not typical of the rash of meningococcemia. The same remarks apply to the rash reported during the interval between the two hospital admissions.

In retrospect, increased importance might have been attached to the history of joint involvement. Such complaints of fleeting pain are not infrequently seen, however, as a non-specific phenomenon in infectious diseases. The various reports of joint involvement in meningococcemia stress the point that there is an "arthralgic" type, characterized chiefly by pain and some swelling but without evidence of suppuration. If, as seems likely, the original complaint, in October, 1945, marked the onset of meningococcemia, one would classify the joint involvement as of the arthralgic type.

Apparently there were no other localizations, and the absence of growth in any of the four blood cultures taken at the first admission indicates that such bacteremia as was present was probably not heavy. It is possible that had the blood cultures been made with special technics later used to recover the organism from the abscessed inguinal gland, positive results might have been obtained. Yet, there appeared to be no reasons clinically at the first admission, to suspect an organism which required special conditions for growth. It has been advocated by some that in any condition where an infection is suspected and no organisms recovered by usual methods, whether it be from blood or pus, the laboratory should employ special anaerobic technics routinely. Such a procedure would be of help in many cases but is obviously expensive and would require more laboratory staff.

The interval from apparent onset of the disease, October 24, 1945 to the time the gland was first noticed, December 21, 1945, was almost two months. Furthermore, she was afebrile during the last ten days of her first hospital stay and during her second admission until after the gland had become fluctuant. During the interim she was reported to have had only a slight afternoon rise in temperature. One can postulate either a continuing meningococcemia of very low degree unaccompanied by significant fever and followed by late localization in the inguinal gland, or, perhaps a seeding of the organisms in the particular gland during the first febrile episode, with a long period of incubation before suppuration took place. Neither explanation is completely satisfactory.

No chemotherapy was given at the first admission and sulfadiazine given on the second admission served only to produce a sensitivity reaction. Since at the time the penicillin was started, the gland had already been opened and was healing, it is doubtful if penicillin played an important role in the child's recovery. It is interesting and important to note, in days when chemotherapy is employed on slightest provocation, that the child's own defense aided by mechanical drainage of an obvious suppurative process, successfully cured the disease.

SUMMARY

A case of isolated suppurative lymphadenitis is reported. The etiologic agent was a meningococcus, Type I. It is believed likely that the lesion was a complication of chronic meningococcemia although positive blood cultures were not obtained.

We wish to thank Dr. Marion Hood of the Bureau of Laboratories, Florida State Department of Health, formerly of the Department of Public Health, Louisiana State University, School of Medicine, for her interest in this case and for her help in writing this report.

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THE WELL CHILD*

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The "well child", at first glance, would seem to be almost too simple a subject for discussion, but more careful perusal reveals its complexities. Our children are the nation's collateral for long term investments. Aside from the purely practical business of admitting that an ounce of prevention is worth a pound of cure, we all agree it is the aim of a democratic society to see that the sick infant or child becomes well and that the healthy infant or child stays that way. Our aim is not to provide husky atomic bomb fodder at some future date, but rather to provide an even start when the children of today have developed to the point they can enter the competitive race of tomorrow.

Synonymous with the "well child" is the "happy child." Certainly no child is completely well who, though sound of body, has a sense of insecurity in his environment. It is the purpose of this paper to review briefly some of the salient principles which foster optimal physical as well as mental growth and development.

PHYSICAL GROWTH AND DEVELOPMENT

The strides made by the biologic sciences within the past few decades have greatly lengthened the average life span. A considerable part of this increase is due to our ability to carry children through the hazards of childhood. Pasteurization of milk was a forward step in this program. Development of scientific infant feeding reduced tremendously the dreaded scourge of cholera infantum, or infantile diarrhea. Reduction in incidence of the contagious childhood diseases, by extension of our prophylactic immunization programs, is common knowledge. Unfortunately, however, there are still some serious childhood diseases for which no protective vaccines have been developed. We are concerned about the recent outbreak of poliomyelitis, yet we know practically nothing about its transmission. Rheumatic fever is another childhood disease of which our knowledge is limited. It is even more crippling than poliomyelitis, although its effects are not as obvious. We can all expect that continued research in these fields will eventually bear fruit. Particularly encouraging are such developments as the adaptation of various strains of poliomyelitis virus to inexpensive laboratory animals, such as cotton rats and white mice. Probably such adaptations will stimulate vital research in this field and will eventually amplify our knowledge of the epidemiology of this disease. Many reports of chemoprophylaxis for group A streptococcal infections have been favorable; these measures may prove to be potent weapons in the control of rheumatic recrudescences.

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*Condensation from an address delivered at the Health Education Workshop, Southeastern Louisiana College, Hammond, La., on September 2, 1946.

Many parents still do not appreciate the practice or wisdom of visiting a physician primarily to keep their child well. An annual physical examination should be urged for every apparently healthy school child. During this visit there should be a comprehensive review of the past medical history, a complete physical examination, such labatory work as may seem indicated, and a frank discussion of all the child's physical and mental problems. It is not difficult to recognize the healthy child. He is alert and active, with clear skin and bright eyes. His tissue turgor is good, his muscles firm, and his body strong. During play periods he participates eagerly and vigorously. He gains continually in size and weight throughout childhood, although growth increases are not constant; there are periods of relatively rapid and slow growth increases, particularly in the preschool period. Isolated comparisons of a child's height and weight with many of the standard charts may have little significance, since some children are inherently larger or smaller than the average. Evidence of a constant falling off or gain from standard curves, as determined by serial "longitudinal" measurements, are much more indicative of abnormality.[1]

The gospel of clean milk and water supplies should be reiterated to lower still further the incidence of typhoid and undulant fever, streptococcal sore throat, dysentery, and other less common milk and water-borne diseases. An extensive educational program has greatly reduced tuberculosis in this country; childhood tuberculosis can be practically eliminated by periodic examination of adults with whom children are in close contact. This is desirable because children are most susceptible and least able to overcome this infection. Intestinal parasitism in children is still a major problem in this region. By education in proper hygienic methods and by careful segregation and treatment of infected individuals these debilitating conditions would assume a position of minor import.

Through school health programs, immunization procedures can be publicized. Every child of school age should have been successfully vaccinated against smallpox and satisfactorily immunized against diphtheria, preferably in early infancy, with supplementary immunization at school entrance. All infants should be immunized with pertussis vaccine and routine mimunization of children with tetanus toxoid is now recommended. There is considerable evidence that many of the multiple antigens (such as combined diphtheria-tetanus toxoid) are satisfactory immunizing agents. Where water supplies are doubtful, typhoid vaccination should be mandatory. It has been my practice to administer a "booster" dose of 1.0 c.c. of diphtheria-tetanus toxoid one year after initial immunization. In addition I apply tuberculin patch tests (until positive) and Schick tests at regular intervals. A suggested immunization plan which is in accordance with the recommendations of the Committee on Therapeutic Procedures of the American Academy of Pediatrics[2] is presented in table 1.

In the field of nutrition the physician should play a leading role. Many parents

TABLE 1
SUGGESTED IMMUNIZATION PROGRAM FOR THE WELL CHILD

Age	Procedure
3 months	Smallpox vaccination — primary reaction
6 months	Pertussis vaccination (with Phase I vaccine) 3 injections
9 months	Combined diphtheria-tetanus immunization 2 injections
18 months	Schick test
2 years	Tetanus toxoid (booster — 0.5 c.c)
3 years	Tuberculin test
6 years	Smallpox vaccination — primary, secondary, or immune reaction Schick test Tuberculin test
9 years	Tuberculin test
12 years	Smallpox vaccination — primary, secondary, or immune reaction Schick test Tuberculin test

have a surprising lack of knowledge as to just what constitutes a child's dietary needs and common advice to "eat a good diet" is therefore often meaningless. Almost daily the pediatrician is consulted by parents who feel that their children need some tonic or vitamin. In most instances the only necessary therapy is an adequate well-balanced diet. In addition the physician can alleviate the anxiety of many parents by simple explanations of the variation in growth and metabolic needs that occur throughout childhood. Stress should be laid on the expected reduction in appetite during the preschool period when weight increases are at a minimum. We are a nation of faddists, and at present vitamins are fashionable. One might say that we are being "vitaminized to death." If we are to believe the advertisements, there is practically nothing that vitamins will not cure. Yet the simple truth of the matter is that no vitamin deficiences will develop in normal children on well-selected, adequate diets.

MENTAL GROWTH AND DEVELOPMENT

Pediatrics was one of the first medical specialties to recognize the importance of preventive medicine. Every child should have a program of medical health supervision and preventive mental hygiene adapted to his individual needs. Although final care rests with the parents, the physician, psychiatrist, and educator must all cooperate and offer them leadership.

Just as the nuclear physicists have far outdistanced the political scientists, so in pediatrics have the objective sciences surpassed subjective ones. The child guidance movement is a mere infant among older branches of pediatrics; one naturally expects such a baby to make errors before it grows to maturity. Handicapped by a cumbersome terminology and overburdened by the necessity of treating physically ill children, physicians initially displayed a good deal of inertia toward mental hygiene. Unduly influenced by the behaviorists, the child guidance movement was ultra-scientific in the early stages. Fortunately, the pendulum is now swinging back to a more common sense, middle-of-the-road position encouraged by the active cooperation of physicians who deal with children.

Not long ago infantile debility, or marasmus, was one of the serious common diseases of infancy. Frequently babies living in hygienic surroundings but without benefit of devotion became marasmic, whereas infants living in poorer homes but with attentive mothers often overcame the handicap of their environment and developed normally.[3] It was the need for affection with its subsequent feeling of security that made the difference. As a result the system of foster home placement rather than institutional care has been developed for infants who have no adequate person to care for them. Hospitals are beginning to recognize this necessity for fondling; several have installed old-fashioned rocking chairs in their infant wards; nurses are instructed to rock infants for periods each day. It is as important to gratify the emotional needs of infants as it is to administer diphtheria toxoid. The latter may prevent disease, whereas the former may avert subsequent equally disturbing behavior problems.

An excellent example of carrying the scientific method beyond the range of sanity is illustrated by some fortunately obsolete technics in the feeding of infants. Babies are more old-fashioned than the "surrey with the fringe on top." A newborn infant of 200 years ago could not be differentiated from a newborn of today—both are babies. As Aldrich[4] has pointed out, the developing child is under the compulsion of two forces; preordained growth drives (physical, emotional, and mental) and the opposing impact of the customs of society. The latter force may arbitrarily dictate to a child how much and when to eat, sleep, or even play. Contributions medical science has made to lowering infant mortality have also inadvertently tended to dogmatize infant feeding in the direction of rigid routines. Such systems have completely neglected emotional needs and individuality of infants, even implying that all babies are born with a "gilt-edged seal" that they are "three or four hour interval feeding babies." Fortunately, most (but not all) infants are able to adjust themselves to being fed "by the hands of the clock." We now realize that "infants are people", each with his or her own inherent characteristics. It has been demonstrated that every baby has certain rhythmic time intervals between hunger pangs. It would seem logical, therefore, to offer feedings at such physiologically rhythmic times, since during the newborn period nursing is obviously one of his greatest satisfactions. An excellent illustration that infants and children have considerable capacity for self-regulation is the oft-cited experiment of Davis[5] on the self selection of food. Fifteen infants of weaning age were allowed to choose what they ate from a variety of natural simple foods essential to health and growth; no comments or suggestions were offered by the attendants. The children rarely mixed their foods, they even ate salt separately. Some of the meals were strange affairs consisting of as high as four or five bananas. One child was reported to eat 10 eggs at a single sitting. Yet the nutritional results over a long period of time (involving some 36,000 meals) were excellent. The group equalled or surpassed conventional standards of growth, were free of digestive upsets, and presented no eating problems. An analysis of each child's food intake over a long period showed that they all were within the limits of scientific nutritional standards. These experiments did not prove that a child can be depended upon to select a proper diet under ordinary circumstances, but only when conditions are favorably controlled, that is, children without preformed likes and dislikes, exposed to natural, unprocessed foods only).

Feeding, however, is only one of many problems in which the inherent characteristics of the child must be adapted to the mores of society. In order to become an acceptable member of society the child must eventually be trained for bowel and bladder control. He must learn to suppress some of his possessive, selfish animal instincts and make his contribution willingly to the group. The negativistic period of childhood should be passed without disrupting the rest of the household. Normal sex drives of adolescence must be kept within the structure of our moral code.

It is the duty of all of us to help each child make these adjustments as naturally as possible and without imposing strict regimentation upon him. I believe this can best be done if we remember that children are all individuals and cannot be compressed into a single mold. In an atomic age, Brennemann's statement[6] seems particularly apt: "The child who presents no conflicts and no deviations from a conventionally ideal behavior is a dud; he will never make a noise in the world."

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SOME CORRECTIBLE ANOMALIES OF THE HEART AND GREAT VESSELS

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With recent advances in thoracic surgery interest in congenital heart disease has shifted from the experimental laboratory to daily medical practice. The purpose of this paper is to review and summarize the background of this subject, to discuss recent clinical advances in diagnosis and treatment of certain congenital lesions, and to summarize results of their surgical treatment.

ANIMAL EXPERIMENTAL BACKGROUND

No attempt will be made to cover this field entirely, but a few interesting papers will be cited. In 1909, Bernheim[1] produced mitral stenosis in an animal, studied resultant changes in circulation, and attempted to determine if correction of the stenosis would restore normalcy. Cushing and Branch[2] probably first successfully used a direct approach to the valve through an incision in the cardiac wall of an animal.

In 1910 Alexis Carrel[3] reported his famous studies on experimental methods of diverting blood during operations on the thoracic aorta by: (1) placing a paraffin tube into the aorta, and (2) lateral diversion through a paraffin tube extending from an incision in the apex of the left ventricle to the descending aorta. Included with the above were descriptions of successful suture of the ascending aorta, end-to-end anastomoses of the descending aorta, and successful graft of a segment of vein preserved in cold storage to an artificially produced defect in the descending aorta. Four years later he described the use of a soft-jawed mass pedicle clamp with which all blood flow to and from the heart could be halted for 2½ to 3 minutes without permanent damage.[4] Employing this instrument he was able to perform a number of experimental operations and to demonstrate a wide range of feasible procedures.

In 1922 Allen and Graham[5] described a cardioscope with an attached knife which could be inserted through a small incision in the myocardium. With later improvements in technic, Allen[6] was able to insert this cardioscope through the left auricular appendage and change mitral stenosis to insufficiency with resultant improvement in pulmonary circulation.

There was a surprising lapse of interest in experimental cardiac surgery for about 20 years. In 1943 Shaw and co-workers[7] used sling sutures about the vena cavae and azygos vein to interrupt blood flow permitting opening of the myocardium and visualization of the valves; he found that operations on valves of the right side of the heart were accomplished with greater ease than those on the left. Harken and Glidden[8, 9] discussed various cardioscopes and described a new instrument providing better visualization than older types.

In 1942 Mandelowitz and Leslie[10] anastomosed the pulmonary artery to the left auricle with results imitating those seen in circulatory derangements typified by the tetralogy of Fallot. Relative and absolute increases in systemic over pulmonary blood flow were observed, but anoxemia occurred only after exercise. Of four dogs surviving anastomosis, only one developed osteo-arthropathy; this animal had an absolute increase in systemic volume and lived long enough for the arthropathy to develop. Since there was no anoxemia in this dog the authors attributed these peripheral changes to increased periosteal nutrition and proliferation.

To maintain life during diminished or arrested pulmonary circulation, Gibbon[11] used a DeBakey pump[12] to withdraw blood from the jugular vein, which was then oxygenated and returned to the aorta via the carotid artery. With this procedure three animals survived complete pulmonary arterial occlusion of 12-18 minutes' duration for 9-12 months.

CLINICAL DIAGNOSTIC AIDS

To familiarize diagnostic procedures Robb and Steinberg[13] in 1939 added the use of a contrast technic employing diodrast given intravenously to permit more accurate roentgenologic study of various circulatory anomalies. Reyes and his associates[14] improved and modified Robb and Steinberg's technic by adapting apparatus to secure simultaneous views of the heart from several angles. They reported that normal and pathologic images could be easily differentiated and described the typical changes in orificial and infundibular types of pulmonary stenosis, patent interventricular septum, transposition of the great vessels, tetralogy of Fallot, and interauricular septal defects. They proposed a special method for demonstrating patent ductus arteriosus: the radiopaque material was injected through a thin trocar in the left brachial artery and passed by retrograde flow into the aortic arch, through the patent ductus arteriosus and into the pulmonary artery.

Stewart and associates[15, 16] used a motion picture camera to follow the course of radiopaque material fluoroscopically visualized.

Increasing experience with such means for demonstrating blood flow should facilitate more accurate anatomic diagnosis and selection of cases suitable for surgical correction.

CONGENITAL DEFECTS AND ATTEMPTS AT SURGICAL CORRECTION

Before entering into a discussion of this phase of the subject, credit should be given to Maude Abbott,[17] whose monograph reviewing and classifying 1000 examples of congenital heart disease did so much to bring order from chaos. Maliner and Borow's[18] clinical modification is, however, more practical, classifying most common types into those with and without cyanosis, realizing that some of the latter under severe stress or strain may temporarily show cyanosis. The most common of the acyanotic group are: (1) interventricular spetal defects of smaller size, (2) patent ductus arteriosus, (3) foramen ovale patency, (4) coarctation of the aorta, (5) subaortic stenosis (very rare). The cyanotic group exhibits such basic lesion as (1) pulmonary stenosis (especially with the tetralogy of Fallot); (2) large septal defects (cor bi- or tri-loculare); (3) transposition of the great vessels.

PATENT DUCTUS ARTERIOSUS

Since this subject has been covered by very competent writers, little space will be devoted to it here. In 1940 Gross[19] became the first in this country to close a patent ductus successfully and much credit is due this noted surgeon for his stimulation of interest in this and related problems.

Burch[20] reviewed practically all of the available case reports prior to March, 1944, and found that the average surgical mortality was 8.5 per cent in uninfected ducti, the rate varying, however, with the surgeon and his experience. He recommended the procedure for all patients with this anomaly and superimposed endarteritis, since more than 50 per cent of such patients have been cured by ligation, whereas this condition was previously hopeless. Touroff[21] also strongly recommended ligation when endarteritis is present. Since the publication of these two papers, however, Dawson and Hunter[22, 23] have reported spectacular results in the cure of this conditin with massive doses of penicillin, when the infecting organism is sensitive to this drug.

Thus it seems likely that at present there are four indications for ligation of the patent ductus: (1) progressive cardiac failure; (2) discernible impairment of mental and physical growth; (3) high pulse pressure and, (4) endarteritis when the infecting organism is resistant to available chemo-therapeusis.

Gross[24] has advocated complete surgical division, when possible, as more satisfactory than simple ligation or ligation and wrapping with cellophane to stimulate fibrosis.

PULMONARY STENOSIS

The heading pulmonary stenosis here is misleading in that it is merely the basic lesion present in a group of defects which have, largely through the work of Blalock and Taussig[25] become amenable to surgical treatment. These authors, quoting Lundsgaard and Van Slyke,[26] cite four factors in the production of cyanosis: (1) height of hemoglobin; (2) volume of venous blood shunted into systemic circulation; (3) rate of utilization of oxygen by peripheral tissues, and (4) extent of aeration of blood in the lungs (most important).

After producing pulmonary stenosis in dogs, the idea of anastomosing a systemic artery to one of the pulmonary arteries in order to increase pulmonary circulation was conceived. Following these experiments, three human patients were operated upon, using in the first case an anastomosis between the end of the left subclavian artery to the side of the left pulmonary artery; in the second case (in which there was a right aortic arch) the end of the innominate to the side of the left pulmonary, and in the third the end of the innominate to the side of the right pulmonary. Ether and oxygen anesthetic was used in the first case, intratracheal cyclopropane in the second and third. Although no marked impairment of systemic circulation to parts deprived of their major arterial pathway was noted, simultaneous dorsal sympathectomy was suggested to minimize this hazard. Following successful transplantations there has been: (1) a pronounced decrease in cyanosis and dyspnea with an increase in exercise tolerance; (2) decrease in erythrocyte count, hemoglobin, and hematocrit; (3) a fall in oxygen capacity and increase in oxygen content of arterial blood, and (4) most importantly, a decided rise in oxygen saturation of arterial blood.

Experiments are continuing and a great deal of attention is being directed to the relative merits of end-to-end and end-to-side anastomoses. Results of side-to-side anastomosis of the aorta to the left pulmonary artery have been found discouraging. The suture method of anastomosis is preferable to Blakemore's vitallium technic[27] as the latter would not permit increase in caliber with growth.

Though the likelihood of thrombosis at the anastomotic site might theoretically be reduced by the use of anticoagulants, such as heparin or dicumarol, attendant risk of hemorrhage has made this issue controversial.

Anomalies in which such transplantations are indicated include those in which the primary difficulty is inadequacy of blood flow into the lungs, such as, tetralogy of Fallot, complete pulmonary arterial atresia, absent or defective right ventricle, truncus arteriosus with bronchial arteries, or even a single ventricle with a rudimentary outlet chamber in which the pulmonary artery is small. Before considering operation the clinician should be sure that there is: (1) roentgenographic evidence of a small pulmonary artery, and (2) clinical or roentgenographic evidence of the absence of pulmonary congestion. In differential diagnosis complete transposition of the great vessels, right aortic arch and the Eisenmenger complex must be considered. Some apparent failures after operation may be due to irreversible changes in the capillary bed of the lungs caused by prolonged deficiency of blood flow.

COARCTATION OF THE AORTA

Since we Americans are a patriotic group, we always prefer to attribute to one of our nationality the honor of originating ideas and methods. It seems, however, that Alex Carrel,[3] a Frenchman, performed the first animal experimental end-to-end suture of the aorta followed by recovery, and Crafoord,[28] a Swede, apparently first operated successfully on two cases of aortic coarctation. The latter clamped and resected the constricted segment and reanastomosed the ends.

Gross and Hufnagel[29] became quite interested in this lesion and reviewed the advantages and disadvantages of procedures such as those advocated by Blalock and Park[30] Carrel,[3] and Blakemore and Lord.[27] After animal experimentation Gross[29] treated two patients according to his own technic which closely resembled that of Crafoord.[28] In all four cases reported as successfully treated by these two surgeons, excellent results have been obtained.

TRACHEAL OBSTRUCTION FROM A VASCULAR RING

Again Gross[31] reported that vascular rings within the mediastinum are of two types: (1) the aortic arch absent from its normal position in front of the trachea, coursing instead behind the esophagus, the pulmonary artery lying in its normal position in front of the trachea but anchored firmly by a patent ductus arteriosus or remnant to the distal part of the aortic arch. Thus the trachea is compressed anteriorly by the pulmonary artery, and the esophagus is compressed posteriorly by the aortic arch. Division of the patent ductus arteriosus or its remnant should give relief. (2) The second type is a divided or split aortic arch, one limb passing posteriorly, then to the left behind the esophagus, and the other coursing to the left in front of the trachea, the two limbs then converging to form the descending aorta. Either or both limbs may rise to major arteries, and the limbs may be of the same or unequal size. The tracheal and esophageal constriction may be relieved in some cases by dividing the ring, while in others this operation is impossible without severing vital arterial pathways.

Gross[31] reports a four month old infant with difficult wheezing respiration since birth, the tracheal obstruction resulting in four attacks of tracheitis of sufficient severity to require hospitalization. Roentgenographic evidence of a vascular ring was found and the suitable operation performed, both types of rings described above being found. The procedure was well tolerated by the patient with marked alleviation of symptoms.

Diagnosis is based, in this condition, upon the demonstration of indentation of the esophagus by barium study, and anterior constriction of the trachea shown by roentgenogram after lipiodol instillation with anteroposterior and lateral films. Only in certain cases does the presence of a vascular ring produce symptoms, but when symptoms are present; surgical correction is indicated.

SUMMARY

Accurate diagnosis followed by carefully planned surgical treatment is no longer an experimental procedure but rather a clinical reality for many types of congenital heart diseases. Animal experimentation relative to the subject is considered, together with recently described diagnostic aids and clinical reports of surgical treatment of certain anomalies.

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SPLENOMEGALY WITH CIRRHOSIS OF THE LIVER COMPLICATING JUVENILE DIABETES

A CASE REPORT WITH SPLENECTOMY

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The following case is reported because of its interesting features and because no similar case has been found in a search of the literature.

CASE REPORT

L. S. B., a white male, aged 13, was referred to me on November 22, 1943 because of generalized edema of the feet, scrotum, abdomen and face. He had been known to have diabetes for the previous two years and had been under medical treatment. His original complaint was polyuria and inability to gain in weight. He had been on a restricted diet but without special supervision. He had been accustomed to taking 10 units of protamine zinc insulin and 15 units of regular insulin mixed in the same syringe before breakfast. At noon he would take 10 units of regular insulin and 15 units of regular insulin before supper. For the previous week he had been troubled with diarrhea, having as many as 15 movements a day. He had been given bismuth preparation which apparently partially controlled the diarrhea. Several days later he developed an acute respiratory infection and the diarrhea returned.

Past History: He had had the usual childhood diseases but had never been sick for any extended period of time. He was always thin and never complained. He had always had an excellent appetite.

Examination revealed a white boy whose skin was sallow. There was pitting edema of both legs extending to the knees. The scrotum was markedly edematous and almost the size of a grapefruit. The prepuce was swollen and he had difficulty in voiding. The skin of the abdomen was tense and showed pitting edema. The hands and face were likewise swollen. There were no petechiae. The temperature was 99, pulse 120, respirations 18. The tonsils were large. There was a post-nasal drip with pus from the right ethmoid. The heart was slightly enlarged to the left to percussion. The sounds were regular and of good volume. There was a loud blowing systolic murmur present. The abdomen was tense and a fluid wave was present. The liver and spleen could not be felt. His weight was 83½ pounds.

Laboratory findings showed the total 24 hour urine output 1100 c.c., clear, specific gravity 1040, acid, no albumin, 6 per cent sugar, 2 plus acetone, no diacetic acid. The urine was negative for indican and showed 1 plus urobilin. Hemoglobin was 55 per cent, red blood cells 2,200,000, white blood cells 4,000. The smear showed basophiles 1 per cent, eosinophiles 1 per cent, myelocytes 1 per cent, juveniles 3 per cent, stabs 9 per cent, segmented 57 per cent, lymphocytes 25 per cent. The sedimentation rate was 35 in half an hour. The blood sugar was 171 mgm. per 100 c.c. of blood; serum protein 6.05. B.M.R. plus 4. The Wassermann was negative. The stool was negative for parasites or ameba.

It was obvious that this was a case of juvenile diabetes complicated by cirrhosis of the liver or chronic adhesive pericarditis associated with the rheumatic heart disease. During the following ten days an effort was made at establishing control of his diabetic condition and while this appeared to be under control he continued to gain in weight. Four days later his weight increased to 94½ pounds. He was given tr. digitalis until therapeutic effect was obtained. Weight loss was slow and on December 10 he was given 500 c.c. of whole blood. There was still no appreciable reduction in his weight. On December 16 he was given 1 c.c. salyrgan-theophyllin intravenously and again on December 20. The urinary output increased to 3000 c.c. From then on he began to lose weight. On January 5, 1944 he weighed 82 pounds. He was receiving 1900 calories with 15 units of protamine zinc insulin plus 15 units regular insulin for breakfast, 20 units regular for dinner and none for supper. With this regimen he continued to maintain his weight constantly and remained relatively free of sugar.

On February 26 his abdomen was found slightly distended and upon examination a large hard spleen was found which was freely movable and extended medially to the umbilicus and inferiorly to the crest of the ileum. In spite of the difficulty of controlling his diabetes and recurring episodes of edema, it was felt that a splenectomy was indicated. He received two transfusions preoperatively and was digitalized.

On March 13, 1944, under gas and ether anesthetic, a left mid-rectus incision was made exposing a large, freely movable spleen which was easily delivered into the wound. All vessels were ligated in the pedicle and the spleen was removed without any difficulty. The liver was slightly enlarged and rusty brown in color. It was hard and showed a diffuse granular appearance characteristic of cirrhosis. He had a normal postoperative convalescence which was afebrile. The wound healed per primum and he was discharged on the seventh day.

Pathologic specimen showed a large spleen measuring 6" x 4" x 3". The edges were round and hard. The splenic notch was present. The spleen weighed 800 grams. The tissue cut with difficulty and the section showed the characteristic fibrosis present in Banti's splenomegaly.

On April 1, 1944 his weight was 80 pounds. His urine was free of sugar. His diet was maintained at 1900 calories and he returned to his preoperative insulin requirements. The liver edge was palpable three finger breadths below the costal margin.

Since his splenectomy he has never shown any edema and has been able to control his diabetes. He remained well until the beginning of 1945, when his diarrhea recurred. In April 1945 he began to lose weight. Stools which were almost watery in consistency were negative for ova and pathogens. Analysis of the stool indicated that the diarrhea was due to a fatty intolerance caused by atrophy of the pancreas. Large doses of pancreatic gland were necessary to control the diarrhea.

DISCUSSION

Juvenile diabetes is not as unusual as formerly believed. These cases, as in adults, are most frequently detected in the course of routine urinalysis. They are most difficult to control because allowances must be made for growth as well as the child's normal desire for sweets.

Splenomegaly is likewise not uncommon in childhood. The splenomegaly of Banti's disease, if there is such a syndrome recognized now, is probably the most frequent type of enlarged spleen. Cirrhosis of the liver at age 13 with ascites and generalized anasarca is, however, very unusual. A diligent search of the literature fails to reveal a similar case.

SUMMARY

A case report of a white male, aged 13, who has had known diabetes for two years and who coincidentally had a splenomegaly with cirrhosis of the liver, is presented. Splenectomy was performed because of the generalized anasarca and ascites without subsequent recurrence.

THE WATERHOUSE-FRIDERICHSEN SYNDROME

WITH A REVIEW OF THE LITERATURE AND AN ADDITION OF THREE NEW CASES FROM THE CHARITY HOSPITAL OF LOUISIANA

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HISTORY

Although Voelcker reported the first presumptive case of this disease in 1894, Andrews in 1906 was the first worker to describe a proved case of fulminating memingococcal septicemia, now commonly known as the Waterhouse-Friderichsen syndrome, in the literature.

In 1911, Waterhouse added one case and reviewed the 15 others in the literature up to that time. Seven years later Friderichsen described two of his own cases and collected 28 cases altogether.

Glanzmann, in 1933, was the first to apply the term Waterhouse-Friderichsen syndrome to this clinical picture. Three years later, Aegerter and Sacks reviewed the literature separately and collected 55 and 64 cases, respectively.

During the following years, among others, Kundstäedter added one case, Moss and Schenken two cases and Lindsay et al. seven cases, so that by 1943 Bernard and Jordan were able to collect 117 cases in the entire literature.

I have attempted to review the literature since that time and have collected 57 more cases: Rucks and Hobson, Potter and Bronstein, Hayes and Whales, Weinberg and McGarvack, Thomas and Leiphart, and D'Agati and Marangoni have each reported one additional case.

Day and Ikeda and Rosenthal have contributed two more cases each, while Strick has described three cases. Marangoni and D'Agati and Bush and Baley have added six new cases whereas Pratt et al. and Cosgriff have both reported four more cases. Finally Martland reviewed 19 new cases of Waterhouse-Friderichsen syndrome.

I have also attempted to review all cases of this syndrome at the Charity Hospital of

Louisiana during the past five years. During this period, only two cases were definitely proved at autopsy and one strongly suspected but not proved case of the Waterhouse-Friderichsen syndrome has occurred at this institution. Since these three cases have apparently not yet been added to the literature, they bring the total of reported cases up to 177, as far as could be determined.

ETIOLOGY

It is commonly agreed that the meningococcus is by far the most frequent causative organism of this disease-syndrome. Various authors believe that it is the offender in from 50 to 70 per cent of all cases. It must, however, be borne in mind that it is not the sole etiologic factor in all cases. The *Streptococcus hemolyticus* is probably the second most commonly isolated organism. Lindsay et al. demonstrated the presence of *Hemophilus influenzae* in both the heart's blood and the cisternal fluid in two of their cases. Other writers report *Staphylococcus aureus, pneumococcus, Bacillus coli, Bacillus pyocyaneus, Bacillus Friedländer* and *Neisseria flavus* respectively as the causative organism in their various cases.

PATHOLOGY

The most constant pathologic condition at autopsy is massive adrenal hemorrhage, occurring in 95 per cent of the reported cases, according to Aegerter. Usually as soon as the renal regions are exposed at autopsy, one is startled by the remarkable appearance of both adrenal glands which are commonly deep purplish red. They retain their normal shape but are increased in size and have tense capsules.

On section, each adrenal gland is the seat of massive hemorrhage which has usually destroyed almost the entire gland. On the other hand, in some cases only minute petechial hemorrhages can be demonstrated. When the bleeding is mild in degree, it is generally stated that the medulla is more involved than the cortex.

Under the hand lense it can be seen that in the middle zone (zona fasciculata) the hemorrhagic areas have a definite pattern of a great many parallel red streaks. In the inner zone (zona reticularis) the hemorrhages often have a lace-like or reticulated appearance.

In this connection it is of interest to note that according to Marangoni and D'Agati the patient's life expectancy is about inversely proportional to the extent of the adrenal damage; that is, the greater the adrenal hemorrhage, the shorter the clinical course and the less likelihood for the development of subsequent hepatic destruction.

Microscopic study reveals an enormous capillary distention of the middle and inner zones of the cortex. There is usually no evidence of inflammation, thrombosis or embolism. In most cases, the outer zone (zona glomerulosa) escapes except where it is involved in isolated, irregular areas. The capsule as well as the surrounding structures only rarely exhibit any evidence of involvement. If proper staining of the tissue is employed, in most cases many clumps of meningococci, or any other etiologic agents, may be seen throughout the cortex.

Nearly every case at autopsy presents an extreme cyanosis and lividity over the face, chest and lower extremities. Ecchymotic and petechial skin lesions are practically constant findings. These often also are seen in the conjunctivae. In about half the cases, small hemorrhages are present in the serosa of the peritoneum, pleura or pericardium. A few reports mention hemorrhage into the mucosa of the gastrointestinal tract. This, according to McLean and Caffey, may account for the abdominal pain and diarrhea sometimes seen. The brain usually shows congestion of the superficial vessels of the leptomeninges. Frank signs of meningitis are characteristically absent. This supports the observation made by Herrick, during the first World War, that most meningococcic infections are bacteremias before there is metastatic localization in the leptomeninges.

Too frequently to be incidental, hyperplasia of the thymus and the other lymphatic structures of the body is associated with this syndrome. For example, in reporting 11 cases in 1901, Little noted that Peyer's patches were much swollen and unusually prominent, "as in the early stage of typhoid", and that in addition the mesenteric glands were enlarged in six and the thymus in two cases.

The other viscera usually show no significant pathologic lesions, with the exception of acute cloudy swelling of the myocardium, the kidneys and the liver and terminal pulmonary edema.

PATHOGENESIS

Various theories have been proposed to explain the selective action of this disease on the adrenal glands. A few of these are as follows:

1. *Embryologic Interpretation*: The skin, adrenal medulla and nervous system all arise from ectoderm and are at the same time the chief organs affected by this disease. It has, therefore, been suggested by some workers that this is not an incidental finding but that the disease possibly may have an affinity for this embryologic layer.

2. *High Incidence of Status Lymphaticus*: The occurrence of the Waterhouse-Friderichsen syndrome, together with findings of status lymphaticus (if such an entity actually exists), has made some authors feel that there is some relation between these two.

3. *Venous Thrombosis*: Another theory states that thrombosis of the central veins in the adrenals leads to capillary distention due to backward pressure and thus causes the hemorrhages into the adrenal parenchyma.

4. *Arterial Thrombosis*: Symmetrical, bilateral thrombosis of the arteries of the adrenals is rather difficult to conceive, and therefore this theory is not widely accepted.

5. *Bacterial Embolism*: Although this is also a possibility, it is, however, not too likely that a great enough number of microorganisms should clump together in the adrenal blood vessels to cause actual obstruction to the blood flow.

6. *Effect of Meningococcic Toxins*: According to this theory, the toxin released by the causative organisms of the disease picture has a direct effect on the capillary endothelium and causes a rupture of its walls with subsequent hemorrhagic extravasations.

The question arises just what role the adrenal gland plays in the production of the Waterhouse-Friderichsen syndrome. Adrenal cortical insufficiency is suggested by:

1. The clinical picture of hypotension, dehydration and vomiting.

2. The laboratory findings of decreased blood sodium, and increased blood NPN, and occasional hypoglycemia.

3. The pathologic findings of adrenal hemorrhage.

Rogoff states that the experimental animal may suffer a loss of both adrenal medullas and one cortex without radically influencing his life and health. However, a bilateral removal of the adrenal cortex invariably leads to the animal's death. In some cases of the clinical syndrome under discussion the extent of the adrenal cortical destruction may be very small. On the other hand, it is well known that only a small portion of the adrenal cortex is necessary for carrying on the essential processes of life. Furthermore, there is only a small number of patients in Addisonian crisis who do not respond favorably to the administration of fluids, electrolytes and cortical replacement therapy. However, patients exhibiting the Waterhouse-Friderichsen syndrome often do not improve on a similar type of treatment. These factors have made some workers believe that hypoadrenalism is not the primary cause of death here, but that the exodus is actually caused by an overwhelming toxicity throughout the body.

Marangoni and D'Agati are of the opinion that the Waterhouse-Friderichsen syndrome consists of two distinct clinical stages: The first stage consists of circulatory collapse and usually leads to death. If the patient survives this stage, the second or hepatorenal stage begins; this is characterized clinically by marked oliguria with azotemia and pathologically by a severe toxic hepatitis associated with glomerular and degenerative tubular changes.

The hepatorenal syndrome was actually first described by Heyd in 1923 to explain certain deaths following biliary operations Wilensky and Colp have advanced the most satisfactory theory thus far to explain this syndrome: they postulate the existence of an initial lesion which affects the hepatic cells primarily and the renal nephron secondarily.

Marangoni and D'Agati believe that adrenal hemorrhage is a predisposing factor in the development of hepatorenal failure and they advance the following hypothesis for this process: Destruction of the adrenal glands by hemorrhage throws the full load of detoxification primarily on the liver and secondarily on the kidneys, both of whose functions have already been depressed from the onset by shock and toxemia. The partially disabled liver is so overwhelmed by toxins that it is unable to perform its physiologic detoxifying function. As a result, the liver undergoes toxic necrosis and a potent tissue toxin is liberated which, together with the unneutralized bacterial toxins, flood the circulatory system. Finally, the kidneys attempt to rid the body of these harmful products of bacteria and deranged liver metabolism by excreting them through the proximal convoluted tubules which, during this process undergo degenerative changes. In addition, glomerular changes of the nephritic type occur.

An interesting sidelight is the fact that the toxins produced by the necrotic liver cells are actually harmful to the uninvolved hepatic tissue. This was proved by Boyce who injected water-soluble extracts of liver tissue from patients dying of the hepatorenal syndrome into dogs by the intraperitoneal route. The dogs all died when strong extracts were used, and hepatic and renal damage could invariably be demonstrated microscopically at autopsy.

MEDICO-LEGAL ASPECTS

Even though it is little recognized as such by the medical profession, the Waterhouse-Friderichsen syndrome often presents a real medico-legal problem. The reason for this is that death in this syndrome is usually extremely rapid (usually within 24 hours after onset of symptoms) and that it is therefore often considered to be of unexplained cause. Martland states that a good number of these deaths have to be investigated because of the suspicion of food or chemical poisoning, foul play or negligence. To illustrate this, a few examples of the résumés of some of his cases follow:

Case No. 1: Adult died in hospital 24 hours after vomiting mushrooms. Mushroom poisoning suspected.

Case No. 2: Adult died two minutes after admission to the hospital and 48 hours after onset of gastrointestinal symptoms. Relatives wanted to sue a "fish-market."

Case No. 3: Adult found dead in bed. Police suspected "mugging" or manual strangulation. Subject dead for two days and body in bad state of decomposition. Subject well when last seen alive.

SYMPTOMATOLOGY

The clinical manifestations of the Waterhouse-Friderichsen syndrome have not been generally recognized. This is probably due not to failure of diagnosis alone but also to the fulminating course of the infection and the scarcity of cases seen by any one observer.

By far the majority of cases occur in patients under one year of age. Lindsay et al. believe that about 90 per cent of all cases are seen in children from six months to nine years. Aegerter states that 70 per cent of the patients have been under two years. During the past few years, there have been many additional reports of this syndrome in adults. This, however, probably does not indicate an absolute increase of this syndrome in adults, but is believed to be due to a better system of case reporting in the armed forces during this war.

The patient typically states that he had enjoyed perfect health until the onset of the disease. It has been found by various workers that the only cases without sudden onset are those complicated by other conditions, which might have altered the characteristic early signs.

The initial symptoms may be quite variable. There may be restlessness, headache and malaise or there may be gastrointestinal symptoms, consisting of abdominal pain, loss of appetite, vomiting and possibly diarrhea. Fever of moderate or extreme degree may be present.

One of the outstanding clinical features is the development of cyanosis. This can usually be noticed first about 12 hours after the onset of the disease, and has frequently been reported even as the first clinical symptom. The cyanosis, being of such a severe nature, is often quite frightening to the relatives and physician alike. It colors the lips and fingernails of the patient dark blue and gives the skin a livid appearance. A few reports mention a peculiar alternation of cyanosis and pallor of the skin resulting in a peculiar mottled appearance.

Soon after the onset of cyanosis petechiae may be noted over almost the entire body. These spread with such extreme rapidity that their development can frequently actually be observed within several minutes. Lindsay, for instance, tells about a patient's mother who told that "the rash was not present when the child left the house for the hospital." The small petechiae present initially usually soon develop into large, violaceous purpuric areas giving the skin a blotchy appearance. Some of these areas are the size of a palm or even larger. They do not disappear on digital pressure. Sometimes these areas undergo necrosis and sloughing later.

When examining the patient at this time, one usually finds him to be desperately ill. The blood pressure is often too low to be determined and the pulse is weak or even imperceptible. The patient is in a state of shock and often is either stuporous or semicomatose. The examiner is impressed with the rapid progression of the disease from hour to hour, which Herrick has so well described by stating that "no other infection so quickly slays."

The extremities generally become cold and the body hot. The patient at this time also becomes incontinent. The respiratory rate greatly increases and respiration often becomes labored. Some patients develop stertorous breathing and typical Cheyne-Stokes respiration. Throughout this period numerous rales can be heard over the chest —at first of the fine crepitant and then later of the coarse, bubbling type. After frank pulmonary edema occurs, the patient usually goes into coma and dies shortly thereafter.

The occurrence of clinical evidence of meningeal involvement is variable. Some authors state flatly that it never occurs in a true Waterhouse-Friderichsen syndrome, while others believe that it is quite common. Many patients complain of violent headache at some time during the disease. The observation of abnormal reflexes, convulsions and various degrees of stiff neck have been recorded.

D'Agati and Marangoni believe that too little attention has been paid in the past to the initial anuria which was a constant finding in their cases. The anuria persisted for 24 to 36 hours and was replaced by a severe oliguria. At no time was adequate renal function reestablished, despite recovery from shock, evidenced by normal blood-pressure readings, the improved character of the pulse and the improved clinical appearance of the patient.

The temperature curve in most cases is of a septic, spiking type, which may range from subnormal to 108 degrees Fahrenheit. Early in the disease the rise in temperature is accompanied by a similar rise in the pulse rate, but as the disease progresses the cardiac rate increases more and more. The cardiac rhythm, however, is only rarely affected, but its quality is generally found to be poor and fluctuating.

There are a good deal of other more rarely encountered signs and symptoms found in the literature, some of which are: muscle flaccidity, low blood sugar, chills, tremor, cough, ankle edema, strabismus and rigidity of the abdominal muscles.

The entire course of the disease is usually unbelievably rapid and, as a rule, the patient expires within 24 to 48 hours after the onset. It can, therefore, easily be seen that it is essential to make the diagnosis as promptly as possible and to institute treatment at once, for if the development of the full clinical picture is awaited, the hope of survival is negligible.

LABORATORY DATA

The occurrence of positive blood cultures has been discussed above. In some cases the organism has been identified in the routine differential blood count. The probability of identification by this method is materially increased if the blood for the count is taken at the site of a purpuric cutaneous lesion. In some cases, leukocytes laden with suspicious diplococci may be observed by mere chance in the Wright's stained blood films. Humphrey and Toomey advocate the utilization of the "buffy layer" smears for gram-staining which permits a concentration of the invaded cells and organisms not possible in other methods. The limitation of the blood smear methods, of course, must be realized in that if the bacteremia is not heavy, it is not as satisfactory as would be a culture, and that in most instances it also lacks the positive identification made possible by culture. However, its use is of great value because by the time the cultural diagnosis is made, the use of chemotherapeusis may have been delayed too long already to be effective.

A mild to moderate leukocytosis with a relative increase of the polymorphonuclear leukocytes is present in most cases. A shift to the left in the cells of the myelogenous series was noted by Bamatter. Occasionally the occurrence of an extreme leukocytosis, with cell counts up to 100,000/cm. mm. has been reported. There is often a moderate reduction in blood platelets. The Leed-Rumpel test is usually positive. Determinations of the coagulation and bleeding time, as a rule, are inconsistent depending on the stage of the disease.

Reports concerning the blood chemistry in the Waterhouse-Friderichsen syndrome are still quite incomplete. It appears, however, that an increase in the blood non-protein-nitrogen, and a decrease of the blood sugar, blood sodium and blood cholesterol levels are fairly constant findings.

It is noteworthy that difficulty is often encountered in these cases in expressing enough blood for counts and smears by the usual needle-prick method and that venepuncture is also often impossible.

Urinalysis frequently reveals varying degrees of albuminuria and a fixation of the specific gravity. Hematuria and cylindruria have also been reported.

Examination of the cerebrospinal fluid reveals no constant alterations, either in the cytologic or chemical components. The causative agents of the disease may or may not be seen on smears. The cell count may be high enough to impart turbulence to the fluid but rarely, if ever, is the fluid frankly purulent. An increase in pressure is noted in a considerable number of patients.

DIAGNOSIS

The clinical picture of the Waterhouse-Friderichsen syndrome is usually characteristic enough for the correct diagnosis to be made. The most important thing is to keep the condition in mind in any critically ill child and to consider it in the differential diagnosis.

D'Agati and Marangoni list a number of points which they consider to be of diagnostic importance: petechial eruption rapidly becoming purpuric; severe shock; facial edema; anuria of 24 to 36 hours' duration; urinalysis showing fixation of the specific gravity, albuminuria, hematuria, or cylindruria; marked leukocytosis; isolation of the meningococcus by culture and, in some cases, by peripheral blood smear; renal failure, as evidenced by elevation of the blood non-protein-nitrogen and creatinine.

TREATMENT

The rational treatment of the Waterhouse-Friderichsen syndrome may be divided into the treatment of shock, toxemia, bacteremia and the administration of adrenocortical hormone substitution therapy.

The occurrence of shock in this disease may be due to a sudden incapacitation of the adrenal glands by hemorrhage. Suggestion for replacement therapy rests on this hypothesis. It may, however, also be due to the overwhelming infection. It is essential to keep these two possibilities in mind in combatting the state of shock.

The patient should be kept at a comfortable temperature. The free use of morphine sulfate and the placing of the patient in the Trendelenburg position is also advocated. Parenteral fluids in quantities of 3000 to 4000 c.c. should be given cautiously over a 24 hour period. The intravenous administration of plasma is recommended by some because of its osmotic effect leading to the elevation of the blood pressure. Normal salt solution should not be given in excess, especially if pulmonary edema is present. The use of adrenalin in the treatment of shock is generally contraindicated, but an occasional 0.25-0.50 c.c. dose has been thought helpful by some clinicians.

Every possible effort should be made to combat the toxemia at as early a date as possible. Lindsay et al. believe that the meningococcus and *Hemophilus influenzae* are by far the two most common etiologic agents. If the invader cannot be immediately identified in a given case they, therefore, assume the presence of one of these organisms. In that case they suggest the routine administration of meningococcal antitoxin intravenously and anti-influenzal serum intramuscularly. The usual dose for the antimeningococcal serum is from 60,000 to 120,000 units within the first 24 hours. All precautions necessary for any form of intravenous serum therapy must, of course, be observed. Antitoxin should not be withheld while possible improvement from chemotherapy is being awaited since prompt neutralization of the toxin may prolong life and give chemotherapy the chance to rid the body of the organisms.

Chemotherapy has its greatest value in combatting the bacteremia. Sulfadiazine, which is the sulfonamide of choice, should be given intravenously at the beginning and its administration should be kept up by either this or the oral route in dosages great enough to maintain a blood level of 15-20 mgm. per 100 c.c. It is advisable to keep the urine alkaline at this time, either by giving 1/6 molar sodium lactate intravenously or by any other means, in order to decrease the chance for drug crystalluria.

It is advisable to augment the sulfonamide chemotherapy by the use of penicillin. Only little information is available on this subject in the literature until now. Weinberg and McGavack suggest the intrathecal administration of the drug initially in doses of 15,000 to 20,000 units, which is to be followed every three hours by the intramuscular injection of 20,000 to 40,000 units. In the very severe cases they believe that the drug should be given intravenously, preferably by the continuous-drip method.

Adrenal cortical hormone is given in the hope of combatting the adrenal failure caused by the hemorrhage. However, its value is still quite questionable. Nevertheless it should be given in adequate doses, as early as possible, and by the intravenous route. Bush and Baley recommend the use of a continuous infusion of 5 per cent glucose in saline, repeated intravenous injections of adrenal cortical extract and an occasional intramuscular injection of desoxycorticosterone.

The use of repeated small transfusions is frequently very helpful in combatting the hemorrhagic tendency. Vitamin K may be used empirically when a lowered percentage of prothrombin can be demonstrated.

Finally, the patient should have the benefit of all possible supportive therapy, such as temperature measures if the fever becomes excessive, sedatives if they seem indicated and oxygen when either cyanosis or respiratory distress are observed.

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CLINICO-PATHOLOGIC CONFERENCE

RALPH V. PLATOU, M. D.*
RALPH G. SMITH, M. D.†
AND
CHARLES E. DUNLAP, M. D.‡
NEW ORLEANS

The following is a clinico-pathologic conference presented to the combined Junior and Senior classes at Tulane Medical School.

PROTOCOL

P. J., a colored female, aged 15 months, was admitted to the hospital at 2:15 p. m. on November 15, 1945 and died at 9:45 p. m. on November 15, 1945.

C. C.: The patient drank one-half glass of kerosene 45 minutes before admission.

P. I.: Mistaking it for water, the mother gave the child a glass of kerosene to drink. She believed the child took four to six ounces, then became unconscious, and within five minutes there was foaming at the mouth, gasping, irregular respirations, and frequent coughing. On the advice of a neighborhood druggist, the mother poured a quarter glass of milk and a teaspoon of castor oil into the patient's mouth, causing more coughing. Coma persisted, but there were no convulsions. Up to this time the child had always been well.

P. E.: T. 101.4°. A very well developed and well nourished colored female infant in deep coma, from which she could not be aroused. There was a distinct odor of kerosene on the patient's breath. No cyanosis or edema were noted. Respirations were rapid but of good quality. Coarse rales were heard throughout both lung fields. Corneal and gag reflexes were intact, and no pathologic reflexes were elicited.

Laboratory: Hemoglobin 10.0 Gm., white blood cells 10,800 with 88 per cent lymphocytes, 8 per cent polymorphonuclear cells, 2 per cent eosinophiles, and 2 per cent monocytes.

Course: Immediately after admission she was given 25 c.c. of 50 per cent glucose intravenously. A generalized convulsion lasting two and one-half minutes occurred without cyanosis. Oxygen was started by a nasal tube, and an infusion of 10 per cent glucose in water was continued for the next six hours at the rate of 50 c.c. per hour.

Four hours after admission a similar dose of 50 per cent glucose was given, followed by subcutaneous injection of sodium sulfadiazine. Five hours after admission a milder generalized convulsion lasted for two or three minutes with no change in the respiratory pattern.

Dyspnea was first obvious at six and one-half hours, with occasional deep inspirations separated by periods of apparent hypopnea. Abdominal distention was poorly controlled by prostigmine. Repeated aspirations of upper respiratory tract yielded only small amounts of mucoid material from the posterior nasopharynx; metrazol and artificial respiration during the last hour of life did not affect the increasing respiratory difficulty.

Dr. Platou: I am sure most of you would believe a case of kerosene poisoning such as this one seems too obvious to require much elucidation in a clinico-pathologic conference. Unfortunately, there are lots of loopholes in our knowledge of effects of such

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From the Departments of *Pediatrics, †Pharmacology and ‡Pathology of the School of Medicine, Tulane University, New Orleans.

poisoning, justifying a good deal of elucidation and suggestions for improving our knowledge.

Kerosene, as you all remember, is a mixture of more or less volatile hydrocarbons, and, as you know, the hydrocarbons have certain properties in common. They act in some situations as narcotic agents, and many of them are of inestimable value in anesthesia. Others act as volatile asphyxiants, and most are chemically inert in the human body. Kerosene, being a mixture coming off at a given stage in the process of distillation, undoubtedly varies in composition from lot to lot and distillery to distillery. There is no valid method used for estimating the degree of absorption or to correlate intoxication, asphyxia, or narcosis with variable amounts of such absorption. There are a few things that we do know: Enough cases have been studied in humans, dogs, and rabbits to prove conclusively that there is no constant effect on the blood morphology or constituents which have been measured in clinical laboratories. In a few cases there is a transient acetonuria; a few cases of methemoglobinemia have been reported, but in larger series this finding has been notably inconspicuous. If one gave experimental animals stated doses of kerosene of known composition by various routes and followed the iodine numbers of the serum fatty acids, he might indirectly relate degree of absorption to observed effects. This has not been done, however, as far as I have been able to determine. Because the narcotic properties of a hydrocarbon increase with its volatility or in inverse proportion to molecular weight or its solubility and because kerosene is made up of a mixture of varying molecular weights, volatility, and so on, it is not surprising to note that its effects in humans as well as in experimental animals have been variable.

When we started our work here, we laid down a rule for our service as far as kerosene is concerned—that no stomach tube should be used. I have no reason to change the rule because of this case. So far as I can gather from experimental work and reported human cases, deaths rarely if ever occur unless there has been aspiration. In animals the lethal dose of kerosene has been upwards of 20 c.c. per kilogram given by stomach tube. Given intratracheally, amounts as little as 0.5 to 0.75 c.c. may be lethal within a very few hours. If the patient has just swallowed kerosene and has none in the tracheobronchial tree, there will usually be only a relatively profound narcosis with recovery. If the kerosene were both ingested and aspirated, the harm has already been done and there is no means of effectively removing the hydrocarbon from the tracheobronchial tree by mechanical means. If the child has not already aspirated, then the passage of a stomach tube with the usual incidental struggling, gagging, and vomiting is certainly a good way to induce aspiration.

All the collected observations in experimental animals and humans certainly justify the contention that the single most important prognostic feature is the amount of hydrocarbon in the lungs; mortality rate is related directly to the amount of aspiration.

There are other effects with ingestion of kerosene, a rather violent gastroenteritis, with nausea, vomiting, and diarrhea being common. These may be masked to some degree by the narcosis. In the tracheobronchial tree there is violent inflammatory edema and congestion of the alveolar capillaries with rupture of a few. I have here a report of a case in a 33 year old seaman who drowned in gasoline, which describes similar pulmonary manifestations. It seems surprising to note that among 133 cases in three series, in addition to a few individual case reports, there have been no good descriptions of pathologic findings in the brain, nor have spinal fluid examinations been recorded; yet many of these cases have symptoms suggesting meningeal irritation.

Many events observed clinically after kerosene ingestion are illustrated quite typically here. Aspiration, as in this child, is evidenced by coughing and gagging immediately after ingestion. There are convulsions in 2 to 3 per cent of the cases. The critical period during which most deaths occur is also well illustrated here—within twelve to fourteen hours. Aspiration in this case was perhaps further encouraged by the druggist's advice to administer milk and castor oil to a child already comatose and unable to swallow; we may see castor oil and milk curds in the lungs in addition to the kerosene pneumonitis. The child weighed 22 pounds, and if the mother's estimate of 140 to 150 c.c. kerosene ingestion is correct, she may have received a lethal dose by ingestion alone but had plenty of opportunity for far more than a lethal dose by aspiration.

Regarding treatment, we are faced with the situation in which the narcosis is perhaps due to a combination of asphyxial and histotoxic anoxia; there are only a few things that can be done. First, let me say gastric irrigation should be avoided, as it enhances the chances of further aspiration. Second, I think with the most obvious feature being deep narcosis, these patients should be stimulated by chemical or mechanical means. Because they are asphyxiated to some degree, oxygen or oxygen-carbon dioxide mixture should be given. For irritative changes in the gastrointestinal tract, demulcents and absorbents should be used. Kerosene itself acts as a violent cathartic agent. While the patient is in coma, of course, extreme care must be used to avoid aspiration, and it is perhaps best to give nothing by mouth until he is revived.

The next point should be prophylaxis against the less frequent deaths beyond the acute stage, employing sulfonamides against the usual pyogenic invaders. If death does not occur in twelve hours, the patient is almost always out of danger except for the possibility of a complicating pneumonia, bronchiectasis, or lung abscess. Sulfonamides should be begun prophylactically as soon as the patient has recovered from the narcotic state, or perhaps earlier if given by parenteral routes. This patient, for example, was given sulfadiazine subcutaneously within four hours after admission. Hypertonic fluids may be useful in combatting the violent inflammatory pulmonary edema. This child had evidences of pulmonary edema and may have had cerebral edema as well. The amount of fluid given this patient may be open to criticism. It was not excessive—50 c.c. per hour for six hours. She also received two 25 c.c. doses of 50 per cent glucose. I suppose in the face of obvious pulmonary edema the parenteral administration of fluid could be criticized; here it was used to promote diuresis and reduce pulmonary edema. I think it was justified.

Breathing was of the Cheyne-Stokes type, deep gasps with intervening hypopnea as commonly seen in metabolic alkalosis and in cases of terminal diseases of the central nervous system.

In summary, I believe that nothing was done in management of this case that deserves serious criticism. The administration of 350 c.c. of fluid was not excessive for this patient; faced with the same situation again, we might wish to modify treatment to include more vigorous use of stimulants until narcosis had passed. With this addition, however, I doubt if the outcome could have been improved.

Dr. Rollings: You mentioned that only a small amount of kerosene was necessary to produce death—less than 1 c.c.?

Dr. Platou: Yes, by aspiration. We have no data about the rates of absorption, but it seems obvious that they, as well as the inflammatory changes, occur most rapidly in the respiratory tract.

Student: What do you think about lack of cyanosis in view of the asphyxia?

Dr. Platou: The only answer I have for that is that the patient was colored, and it is often difficult to detect cyanosis in the colored race.

Student: Do you think aminophyllin would have any effect on such narcosis?

Dr. Platou: I doubt it.

Dr. Ray: How easy is it to get an infant of this age to drink kerosene?

Dr. Platou: Surprisingly easy, apparently.

Dr. Dunlap: Dr. Ralph Smith has been kind enough to come in to take part in the discussion of this case.

Dr. Smith: Everything I have to say has already been said, so I will take little time. In these cases gastrointestional manifestations, which are not usually serious, are often experienced. Patients who can describe their symptoms complain of burning pain in the stomach, which may or may not be severe. There may be nausea and vomiting, but it is my impression that vomiting does not always occur. Further irritation of the gastrointestinal tract may cause diarrhea. These effects are probably unimportant as a cause of death.

On absorption, there are actions on the central nervous system. Early, there may be intoxication, more commonly seen with lower boiling point hydrocarbons than with kerosene. This recembles alcoholic intoxication, including delirium and depression of the respiratory center. Occasionally pryexia occurs. There are a few cases recorded with temperatures of 105°F. or 106°F. This patient showed convulsions, which are not uncommon but in the majority of cases they do not appear.

The heart is sometimes affected, as indicated by a rapid, weak pulse. Whether this is caused by a direct action on the heart or is secondary to anoxia, I do not know. I have no explanation for the cherry color of the blood. Methemoglobin has been described but is not a constant finding.

As Dr. Platou has already emphasized, aspiration greatly increases the toxicity of kerosene. In dogs it is from ten to twenty times as toxic administered intratracheally as it is when administered by stomach tube. In one report of 65 cases of kerosene poisoning with six deaths, in all fatalities there was lung involvement, which was assumed to mean aspiration. Of the 59 recoveries, there was pulmonary involvement in 32 per cent. Whether it is possible to have pulmonary involvement without aspiration, I do not know. I think it is much more probable that this would take place with lower boiling point hydrocarbons, such as gasoline or benzene. Kerosene has a boiling point of 150° to 300°C., so that it is not very volatile and the amount excreted by the lungs would not be very great. Gasoline would be excreted by the lungs to a greater degree and would more probably produce lung changes without aspiration.

I am not sure of the cause of death in kerosene poisoning. Probably the best guess is depression of the respiratory center. Again this is undoubtedly contributed to by local irritation of the lungs, causing inhibitory effects on respiration. Aside from any specific effect on the center it is very easy in animals to produce death by putting liquid in the lungs. Fluids going into the trachea instead of the esophagus often cause immediate death. This cannot be entirely due to blocking of the lungs, because it does not take that much fluid. Reflexes probably play a role.

Gastric lavage involves some danger, as Dr. Platou has stated. However, it has not always been considered wise to omit it. In this series of 65 cases which I mentioned, the authors advised gastric lavage, and it was carried out in almost all cases in spite of the fact that they realized that kerosene is much more toxic when aspirated and also that aspiration may take place as a result of passing the stomach tube. They stated that administration of emetics was preferable because it avoided aspiration. Again I have nothing to add as far as lethal dose is concerned. In most cases the amount taken was not known. One-third of an ounce or less has been reported as fatal. Dr. Platou mentioned one case in which 750 c.c. was taken without death. McNally reported one in which a liter was taken with recovery. The variable tolerance may be explained by differences in the rate of absorption and corresponding differences in concentration in the blood stream and brain at any one time. The toxicity of kerosene absorbed from the gastrointestinal tract may be further modified by metabolic changes in the liver, but that is purely speculative.

Dr. Dunlap:This child at the time of autopsy was well developed and adequately if not excessively hydrated. There was edema of both feet and slight subcutaneous edema. When the body was turned over, fluid ran from the nostrils. There was not an excessive amount of fluid in the pleural cavities.

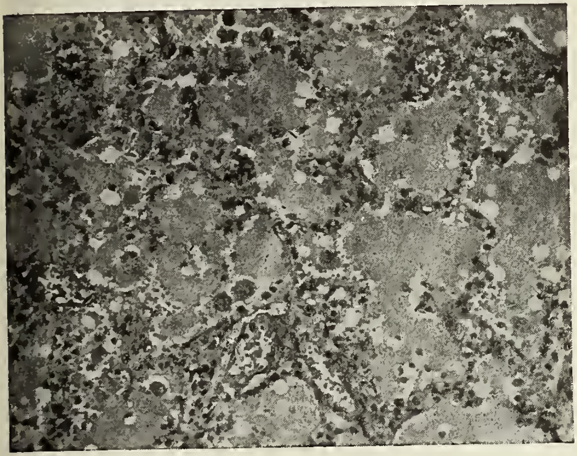

Fig. 1. Photomicrograph of a characteristic portion of the lungs. The alveolar spaces are completely filled with edema fluid except for a few minute bubbles of air. Occasional macrophages have wandered out into the edema fluid. The alveolar walls and other tissues of the lung show no evidence of serious injury.

The lungs were very wet and frothy, and fluid poured from them when they were cut. There was no crepitant lung tissue present except for scattered, small patches; the lungs were thoroughly water-logged. Very little of note was found elsewhere in the body. The stomach was distended with gas and contained about 50 c.c. of white material which looked like milk. There was no gross evidence of injury or hemorrhage in the gastrointestinal tract. The odor of kerosene was easily detected in the contents of the stomach and in the small and large intestines and also in the lungs. Although this is a crude test, I believe it is reliable. The liver showed no gross change except for its yellow color. Special stains revealed a moderate amount of fat in the liver cells. A child dying of almost any disease might have as much fat as was seen here. The heart was not abnormal except for thickening of the endocardium, particularly in the left atrium. Dr. Arey made a careful examination of the brain with routine stains and found no abnormalities. The spinal cord showed no changes. The kidneys showed some degeneration of the epithelium of the convoluted tubules and diffuse hyperemia. The quantity of fluid administered to this child is not clear to me; Dr. Platou may have some inside information, but it is difficult to tell from the chart how much the child received. An infusion of "1000 c.c." was started shortly after admission, and I cannot tell from the chart when it was discontinued. Another "1000 c.c." was started five hours before death and again we do not know over what period of time or at what rate it was given. We have no clear evidence that fluids were administered in excess but we do know that at the time of death the child had massive edema of the lungs and moderate edema of the feet. I believe it is fair to say that the child had adequate fluid and probably too much.

In my opinion this child died of pulmonary edema rather than from any cerebral effects of kerosene poisoning. Lungs that contain almost no air at all and are filled with fluid give us an adequate cause of death without seeking other causes. I do not know the amount of parenteral fluid given, but whatever it was I doubt that it helped the patient and it certainly could have contributed to the development of pulmonary edema.

I do not know of any effective specific treatment for kerosene poisoning but from what has been said by Dr. Platou and Dr. Smith it is clear that control of pulmonary edema might save a good many lives. An article by Hardy and Barach* in the June second issue of the Journal of the American Medical Association discusses the effectiveness of positive pressure respiration in the treatment of pulmonary edema. I would like to read a brief excerpt from their article in order to support a suggestion that positive pressure respiration might have been used to advantage in the case we are discussing today.

"Carlisle reported that the administration of oxygen with the mask which provided pressure in expiration was a most effective method of treating and preventing pulmonary edema due to irritant gases. Rovenstein described a case of irritant gas poisoning in which acute pulmonary edema was successfully treated by the Meter mask, which provided positive pressure in expiration. Segal has also reported the favorable effects of positive pressure respiration in acute pulmonary edema.

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*Hardy, G. C., and Barach, A. L.: Treatment of pulmonary edema due to chlorine gas, J. A. M. A., 128:359, 1945.

"The physiologic basis for employing positive pressure in the treatment of irritant gas poisoning is in the main the application of a direct physical pressure on the external surface of the pulmonary capillaries. Approximately 50 per cent of the applied intrapulmonary pressure is lodged on the capillary wall, the remainder being taken up by the elasticity of the expanded lung. Although positive pressure in both cycles of respiration is a more effective method, the application of positive pressure during expiration alone is simpler."

Positive pressure respiration is relatively safe and may do a great deal to relieve pulmonary edema. Since in kerosene poisoning, survival for twelve hours often means permanent recovery, control of acute pulmonary edema can be a life-saving measure. Lesser, Wiener, and McCoy report that the important pulmonary changes in kerosene poisoning are edema with superimposed pneumonitis. The autopsy findings in this case are in keeping with their observations.

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CHANGES AT LOUISIANA STATE UNIVERSITY, SCHOOL OF MEDICINE

When Dr. Chester Stewart became Director of Pediatrics at L.S.U. in the summer of 1941, the pediatric group in the South was much enriched and improved by the accession of this modest, capable man, who speedily endeared himself to all with whom he came in contact. Dr. Stewart was strictly the product of the Middle West, having received his training in the great state universities of the northern Mississippi Valley. He was keenly interested in research, particularly in the field of tuberculosis in children. It is not generally known that Dr. Stewart had an exceptionally broad training aside from pediatrics, including both anatomy and pathology. In anatomy he was so well qualified that when an emergency made it necessary to name an Acting Head of the Department of Anatomy at L.S.U. there was no hesitancy in asking Dr. Stewart to assume this added responsibility. All this was accomplished although Dr. Stewart's health was by no means good, much as his appearance belied this. A long standing cardiac condition unfortunately terminated the fruitful and promising career of this valuable man on February 8, 1946.

When the authorities of L.S.U. began to cast about for a worthy successor to Dr. Stewart there was found among pediatricians and medical educators an almost unanimous opinion that the best possible pediatrician to fill the vacancy would be Dr. Myron E. Wegman, a young man connected with Cornell and Johns Hopkins in academic work and with the New York City Department of Public Health as director of training and research in child hygiene and director of school health.

In contrast to Dr. Stewart, Dr. Wegman is a product of the northeastern universities, College of the City of New York; Yale, where he took his degree in Medicine; and Johns Hopkins, where he took a graduate degree. While Dr. Wegman's interest is primarily in clinical pediatrics he may be expected to stress the preventive aspects of the subject—a field to which he has devoted so much time and interest. We may safely predict for Dr. Wegman a brilliantly successful career in his new field of endeavor.

The pediatric department is also fortunate in that the new dean at L.S.U. is a pediatrician. Dr. Vernon W. Lippard, recently associate dean at the College of Physicians and Surgeons of Columbia University, took both his undergraduate and medical degrees at Yale and received his pediatric training in New Haven and New York. During the war he served in the South Pacific and held the rank of Colonel on return to civilian life. Although administration of the affairs of the School of Medicine will be his chief responsibility, Dr. Lippard will participate actively in the work of the Department of Pediatrics and will bring to it the benefit of his outstanding experience in research and teaching.

G. W. McC.

ACCIDENTAL POISONING OF INFANTS AND CHILDREN

Although no individual physician encounters very many examples of acute poisoning in his daily work, the magnitude of this problem in hospital practice is great. We have been impressed by the large number of infants and children dying in our hospitals each year as a result of such avoidable accidents as aspiration of kerosene, ingestion of roach paste, or corrosive esophageal strictures due to lye.

All would agree that prevention is the only logical solution to this important problem. Physicians must necessarily assume a leading role in any effective continuous educational program for this purpose. Specifically, any physician caring for children should count among his responsibilities the duty of warning parents of the common sources, nature, and effects of at least the most common poisons. Just as mass immunization programs for infants and preschool children have materially reduced hazards from many common communicable diseases in these age periods, so can conscientious propaganda reduce morbidity and mortality from such even more preventable causes.

Effective therapy in many true emergencies is often delayed by failure to identify promptly particular toxic agents or by injudicious hasty measures so freely advised by well-meaning neighbors and friends. Recommendations for immediate treatment of particular poisonings posted in the accident rooms of many large hospitals are often outdated and sadly in need of revision.

We should like to make this an editorial plea that all practicing physicians review their readily available references concerning most recent advances in therapy and form appropriate committees to prepare up-to-date recommendations for guidance of their own hospital staffs. Having a background of experience with the consequences of common poisonings, pediatricians should be best equipped to institute sensible campaigns for prevention and proper therapy.

M. L. W.

PEDIATRIC NUMBER

This is the second December issue of the Journal that has been devoted exclusively to pediatric subjects. The first pediatric number was made up of contributions from the Departments of Pediatrics of Tulane and Louisiana State University Medical Schools. At that time the late lamented Dr. Stewart, who was Professor of Pediatrics in his school, wrote an editorial in which he expressed hope that articles for later issues would be submitted by physicians who are particularly interested in diseases of children and of childhood. This second pediatric number has fulfilled the wish of this teacher of pediatrics. Merely taking the first article in this journal, it will be seen that it is an outstanding contribution by a group of pediatricians from Shreveport. It is a truly splendid article in every respect and a superb study of infectious hepatitis. There are other papers as well that have come from men who are not connected with the Departments of Pediatrics in the medical schools which will merit reading and attention.

It is believed that the publication of the specialty number has helped to incite interest in the particular specialty of pediatrics. It might be that other specialties should have special numbers and that, in this way, the general practitioner would feel stimulated to report on some of his interesting cases or an unusual case and that the specialists themselves might also be glad and willing to contribute and to make possible a specialty number.

WHAT ARE THE FUNCTIONS OF A CANCER DETECTION CLINIC?

The development of two cancer detection clinics in New Orleans, one at Tulane and one at Louisiana State University, makes it necessary that we, as physicians, all know and understand the functions of such a clinic and our own relationship to it.

Cancer detection clinics are for *presumably well* individuals. Those patients who have symptoms are expected to consult their own physicians about their complaints. If the physician then suspects the presence of a malignancy, the patient is *not* to be sent to the detection clinic, for the function of the clinic has been fulfilled as soon as a physician suspects the presence of cancer. If the physician seeks aid in final diagnosis, the patient should be sent to another physician, who, in the opinion of the patient's physician, is capable of rendering help through special technics, or to a cancer diagnostic clinic, if one exists in that area and the referring physician elects to use it.

When the detection clinic suspects cancer, the patient is sent back to the referring physician or to a diagnostic clinic, so that biopsy can be done. Biopsies are not done in detection clinics, according to the regulations of the American Medical Association.

Because cancer detection clinics do not presume to diagnose already suspected malignancy, no emergency service has been set up for seeing patients who are suspected of having malignancies by signs or symptoms. Patients are examined in order, as they present themselves, and, as a result of the interest aroused in cancer, one of the clinics is at present about seven weeks behind in appointments. No admitting service is provided to note immediate needs of patients, for presumably there are none.

Physicians, therefore, must know and understand the functions of such detection clinics, so that patients requiring immediate care are not erroneously placed on such waiting lists. The admitting personnel of the detection clinics has done a good, but probably not a perfect, job of keeping this relationship straight. A thorough understanding by the physician is also necessary in order that he, as well as his patients, may obtain the maximum benefits from such clinics.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 ANNUAL MEETING PROGRAM

At the call of the President and in accordance with instructions of the Executive Committee, a meeting for consideration of plans for the scientific program for the 1947 meeting was held in the office of the Society on November 21. Seventeen doctors, including the President, the Secretary, Chairman of the Committee on Arrangements, members of the Committee on Scientific Work and Committee to Study Rearrangement of Annual Meeting Programs and Chairmen of Scientific Sections were present.

After considerable discussion motion was made and carried that the general plan as outlined at the 1946 meeting of the House of Delegates be followed, that a clinico-pathologic conference be held on the first night and three or four symposia on the second day, with half hour periods for questions, and the last day devoted to special papers; leaving to the general chairman in charge of the program arrangement and number of symposia. It was the opinion of the doctors present that if the executive officers see fit a meeting of the House of Delegates could be held on Sunday and continued on Monday morning and a symposium held in the afternoon on Monday.

It was brought out in the discussion that the special papers to be presented on the last day would include papers secured by the chairmen of the various sections, these papers to be presented either before special groups or the general membership, as the sectional chairmen may indicate.

The President, subsequent to the meeting, appointed Dr. Donovan Browne to serve as general chairman of the scientific program for 1947. It was brought out in the meeting that the general chairman should notify the chairman of each section concerning subjects selected for the symposia so that essayists may be selected accordingly. It was felt that special subjects could be rotated, to include one or two each year.

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SPECIAL REQUEST TO MEMBERS

Recently every physician licensed to practice in the State of Louisiana was mailed a brochure, explaining the new prepayment surgical and obstetrical care plan, known as Louisiana Physicians Service, Inc., sponsored and approved by the Louisiana State Medical Society. Accompanying the booklet was a Participating Physicians' Agreement with a letter requesting the doctor to review the booklet, sign the card agreeing to participate in the prepayment surgical plan and support the Society's efforts to defeat socialized medicine by cooperating and making this undertaking a success.

The enrollment of Participating Physicians is in charge of Dr. A. V. Friedrichs, Chairman of the Council on Medical Service and Public Relations.

A review of the participating physicians cards as of November 15, revealed approximately thirty (30%) per cent of the doctors signed the agreements and agreed to cooperate with the Society in the promotion of the prepayment plan. Some have written to the office explaining they were in fields other than surgery and obstetrics, and therefore not covered by the plan. It is the ultimate desire of the Society to include every field of medicine in the plan; therefore, we again ask each doctor to sign the participating physicians agreement even if at this time he can receive no benefit from the plan. It is the Society's desire to be able to report one hundred per cent participation by the doctors.

This is your plan. What you do with it, and about it, will determine the extent to which it will succeed. If you study it, and acquaint yourself and your patients with it, and use it properly, it will help you, help your patients, and make Federal compulsory medicine unnecessary. If you ignore it, abuse it, or fail to participate in the plan, you will be doing all you can to make Federal control of medicine a certainty.

Last month's issue of the New Orleans Medical and Surgical Journal contained a complete description of the plan including the schedule of fees payable for services performed. It is true some of the fees are low and others need adjustment. We acknowledge this with the statement that as soon as experience is gained and reserves established adjustments in the fee schedule will be forthcoming to increase the benefits.

If you have not received the material explaining the plan in the mail, please refer to the last issue of the Journal or write to Louisiana Physicians Service, Inc., Room 103, No. 1430 Tulane Avenue, New Orleans, for additional information.

For those who did not receive, or have misplaced this participating physicians' agreement, a copy of the agreement accompanies this article.

Please cooperate with the State Society by signing the following participating physicians' agreement and return it to the Society's office.

PARTICIPATING PHYSICIANS' AGREEMENT

I, the undersigned, a Doctor of Medicine licensed by the Louisiana State Board of Medical Examiners, in consideration of being accepted as a Participating Physician of Louisiana Physicians Service, Inc. herein termed the Corporation, do hereby agree with the Corporation as follows:

I will perform the surgical and/or obstetrical services specified in the Certificates issued by the Corporation for an individual subscriber whose annual income does not exceed $2,000.00 or to a family group subscriber whose annual income together with the income of his spouse and/or family members, does not exceed $3,000.00, at the rates of compensation set forth in the Schedule of Benefits, and will make no charge to the subscriber for such services. In the event services are rendered to an individual subscriber whose annual income exceeds $2,000.00 or to a family group subscriber whose annual income, together with the income of his spouse or family members exceeds $3,000.00, I will accept the indemnity set forth in the Schedule of Benefits as full payment of liability from the Corporation, for services rendered, and any difference between the indemnity paid and my usual fee may be charged to the subscriber.

Thirty (30) days' written notice of termination of this agreement be given by either party.

There shall be no change in the usual patient-physician relationship except in the manner of payment for services under the Certificate and I remain free to act in relations with my patients in accordance with the custom in ordinary practice.

Accepted and Agreed at New Orleans, Louisiana: **Louisiana Physicians Service, Inc.**	Signed: ……………………………………M. D.
Date……………………………………	Address……………………………………
By……………………………………	……………………………………

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TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

TRANSACTIONS OF THE ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Dec. 9 Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
Dec. 11 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.
Dec. 12 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Dec. 13 Executive Committee, Hotel Dieu, 8 p. m.
Dec. 16 Hotel Dieu Staff, 8 p. m.
Dec. 17 I. C. R. R. Hospital Staff, 12:30 p. m.
Baptist Hospital Staff, 8 p. m.
Charity Hospital Medical Staff, 8 p. m.
Dec. 18 Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
The Orleans Society of X-Ray Technicians, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
French Hospital Staff, 8 p. m.
Dec. 26 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.
Lakeshore Hospital, 8 p .m.
Dec. 27 L. S. U. Faculty Club, 8 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Jan. 6 Board of Directors, Orleans Parish Medical Society, 8 p. m.
Jan. 7 Orleans Parish Radiological Society, 7:30 p. m.
Eye, Ear, Nose and Throat Staff, 8 p. m.
Jan. 8 Woman's Auxiliary, Orleans Club, 3 p. m.
Touro Infirmary Staff, 8 p. m.
Jan. 9 Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Every Thursday, Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

RADIO PROGRAM

The following "Health Discussions" were presented over radio station WDSU during the month of November:

November 4—The Eye, Dr. John Gooch.
November 11—Epilepsy, Dr. J. A. Colclough.
November 18—Stomach Ulcers, Dr. Jules Myron Davidson.
November 25—Deafness, Dr. Mercer G. Lynch.

Our radio programs for the year 1946 came to a close with the November 25 presentation. Time given to us by station WDSU has been withdrawn because of other commitments.

NEWS ITEMS

The following members of the Society attended the Fourteenth Annual Meeting of the Central Association of Obstetricians and Gynecologists in Chicago, September 12-21:

Drs. W. D. Beacham, John S. Herring, C. Gordon Johnson, Walter Levy, Harry Meyer, and Thomas B. Sellers.

At this meeting Dr. Beacham presented a paper on "The Management of Large Uterine Myomata with a Report of a 55 Pound Tumor."

Dr. Hunter L. Harang attended the Twenty-first Annual Congress of Anesthetists in New York, September 9-13. Dr. Harang presented a paper on "A Combination of Sodium Pentothal and Nitrous Oxide for Oral Surgery."

Dr. B. Bernard Weinstein attended the meeting of the Alabama State Association of Obstetricians and Gynecologists in Birmingham, October 17. At this meeting Dr. Weinstein discussed a paper on "Sterility."

CHANGES IN MEMBERSHIP ROLLS

During the month of October the following changes took place in our membership rolls:

Dr. H. B. Alsobrok, an active member, was elected to honorary membership.

Two associate members (Drs. Mayo L. Emory and N. Leon Hart) were transferred to active membership.

One active member (Dr. Jos. M. Montagnet, Jr.) and one member recently returned from military service (Dr. Emile A. Bertucci, Jr.) were elected to inactive membership because of their temporary removal from New Orleans.

Dr. Gilbert Balkin, recently separated from military service, was removed from the rolls because of his removal to Denver.

CHANGES IN ADDRESS

Dr. Irvin Cahen, 3705 Prytania Street

Dr. J. A. Colclough, 406 Marine Building.

Dr. Wilfred Finkelstein, 1320 Foucher Street.

Dr. Samuel Karlin, 204 Medical Arts Bldg.

Dr. Louis K. Levy, 3705 Prytania Street.

Dr. Margaret A. Maynard, 1318 Foucher Street.

Dr. Joseph Stamm, 1427 Delachaise Street.

SEPARATED FROM MILITARY SERVICE

The following physicians were recently separated from military service:

Dr. Emile A. Bertucci, Jr., St. Elizabeth's Hospital, Bogalousa, La.

Dr. Joseph L. DiLeo, 1903 So. Dupre Street.

Dr. Jack R. Hays, Charity Hospital.

Dr. Gilbert Balkin, 1003 Republic Building, Denver, Col.

Dr. Warren Hebert, 810 Hibernia Building.

LIBRARY NEWS

Members of the Society who read Italian will be interested to know that the valuable files of the Italian journal Policlinico have been presented to the Matas Library by Dr. Rudolph Matas on the occasion of his 86th birthday.

This journal is published in three sections, (Sezione Chirurgica, Sezione Medica, and Sezione Pratica), and contains publications representative of the best medical thoughts of Italy for the past 50 years.

NOMINATED FOR OFFICE

At the regular meeting of the Society held Monday, November 11, 1946 the following nominations for Officers of the Orleans Parish Medical Society for 1947, and Delegates to the Louisiana State Medical Society for 1947-1948, were handed to the Secretary in accordance with the By-Laws:

President-Elect—Dr. Max M. Green. Endorsed by Drs. I. Tedesco, Jules Myron Davidson, N. J. Tessitore and Daniel J. Murphy.

First Vice-President—Dr. J. Kelly Stone. Endorsed by: Drs. Donovan C. Browne and George Hauser.

Second Vice-President—Dr. C. J. Brown. Endorsed by: Drs. Joseph P. Palermo and Max M. Green.

Third Vice-President—Dr. J. O. Weilbaecher, Jr. Endorsed by: Drs. Wm. H. Roeling and Eugene H. Countiss.

Secretary—Dr. N. J. Tessitore. Endorsed by: Drs. N. J. Chetta and C. Barrett Kennedy.

Treasurer—Dr. Boni J. DeLaureal. Endorsed by: Drs. Eugene H. Countiss and C. Grenes Cole.

Librarian—Dr. Eugene H. Countiss. Endorsed by: Drs. Donovan C. Browne and Eugene B. Vickery.

Additional Members Board of Directors—Dr. E. L. Leckert. Endorsed by: Drs. J. G. Menville, L. A. Delery, L. J. Gehbaeur and Jos. J. Ciolino. Dr. Charles B. Odom. Endorsed by: Drs. Val Fuchs and E. J. Richard.

DELEGATES TO THE LOUISIANA STATE MEDICAL SOCIETY

Dr. Ruth Aleman. Endorsed by: Drs. N. J. Chetta and Vincent P. Blandino.

Dr. Boni J. DeLaureal. Endorsed by: Drs. Edwin L. Zander and Vincent P. Blandino.

Dr. P. Graffagnino. Endorsed by: Drs. B. Hauser, Max M. Green and John F. Oakley.

Dr. Max M. Green. Endorsed by: Drs. Frank Chetta and Wm. H. Roeling.

Dr. George Hauser. Endorsed by: Drs. John T. O'Ferrall and E. J. Richard.

Dr. John Menville. Endorsed by: Drs. Jules Myron Davidson and N. J. Tessitore.

Dr. Waldemar Metz. Endorsed by: Drs. E. L. Leckert and L. S. Charbonnet.

Dr. Daniel J. Murphy. Endorsed by: Drs. Ignatius Tedesco and H. Ashton Thomas.

Dr. J. D. Rives. Endorsed by: Drs. Joseph P. Palermo and C. J. Brown.

Dr. Robert Sharp. Endorsed by: Drs. John Menville and Donovan C. Browne.

Dr. Edmond Souchon. Endorsed by: Drs. Eugene H. Countiss and Pascall L. Danna.

Dr. N. J. Tessitore. Endorsed by: Drs. E. A. Fatter and L. C. Delery.

Dr. Eugene B. Vickery. Endorsed by: Drs. John F. Oakley and Edward Matthews.

Since there is no opposition the Secretary will cast the ballot for the Society on Saturday, December 14, 1946.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

RAPIDES PARISH MEDICAL SOCIETY

The Rapides Parish Medical Society held its regular monthly meeting at the Hotel Bentley on Monday, November 4. Speaker of the evening was Dr. H. Whitney Boggs of Shreveport who spoke on the interesting subject of new developments in the treatment of the allergic patient of particular interest to the general practitioner.

SIXTH DISTRICT MEDICAL SOCIETY

The Sixth District Medical Society held its Fall Meeting in Plaquemine, Louisiana, on Thursday, November 7, 1946.

Dr. Charles J. Bloom read a paper on "Recent Advance in Pediatric Practice". Drs. Gilbert Anderson and Andrew Friedricks held a "Clinical-Pathological Conference". Dr. Will E. Barker Jr., President, presided at the meeting. There was a very good attendance.

ASCENSION PARISH MEDICAL SOCIETY

A regular meeting of the Ascension Parish Medical Society was held at the Gonzales Hospital on Thursday, October 31, at 7:30. The following were present: Drs. Henry Folse, Percy Le Blanc, Earl Schexnayder, Bertha Stokes, D. C. Brumfield, E. S. Kyes, Guy St. Amant, Alvin St. Amant, Myer Epstein, Elsie B. Cassidy, Maurice McConnel, and G. L. Gaudin, Dr. Silverman the guest speaker, and Dr. Alan Leslie, of New Orleans.

It was decided by the society that the regular meeting in December be a joint meeting with the election of officers and scientific program. The society went on record as endorsing the action of the State society in regard the E.M.I.C. program. Drs. Maurice McConnel, Alvin St. Amant, and Elsie B. Cassidy were voted in as new members of the society and Drs. Silverman and Leslie as honorary members.

A paper entitled "The Unstable Colon" was presented by Dr. Earl Schexnayder. Drs. Percy Le Blanc and J. M. McConnel gave brief discussions of this paper and Dr. Silverman, a noted authority on the subject, discussed the paper.

Following the adjournment a most delicious steak supper was served at the new Airliner Restaurant.

G. L. Gaudin, M.D., Sec. Treas.

THE TOURO MEDICAL STAFF MEETING

The regular monthly meeting of the Medical Staff of Touro Infirmary was held Wednesday, November 13, at which time the following program was presented:

First came a Clinico-Pathologic Conference led by the Department of Pathology, the clinical discussion of the case being made by Irving J. Glassberg.

The next scientific presentation was a paper by Drs. E. Burns and W. E. Kittredge on ureterocutaneous anastomosis.

The program was concluded by a 15 minute sound film on sulfathalidine in the treatment of ulcerative colitis.

The Program Committee of the Staff consisted of Drs. Sidney Copland, Oscar Blitz, and Harry Meyer.

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE (SOUTHERN SECTION)

Dr. Chapman Reynolds has sent out notices from the Department of Pharmacology of the L. S. U. School of Medicine to the effect that the next scientific meeting of the Society will be held in Birmingham on December 7. He suggests in his notice that car-pools be established in Memphis, Atlanta, and New Orleans so that members might drive to this meeting together.

MEDICAL DIVISION OF THE CHARITY HOSPITAL VISITING STAFF

The regular monthly meeting was held at the Auditorium of the Charity Hospital, November 19. The program consisted of a paper by Dr. N. Burnstein on Boeck's Sarcoid. The discussion of this paper was opened by Dr. Chester Fresh. A medical case was presented by Dr. C. B. Luikart which was discussed by Dr. Seabury. There was a dermatology case presentation by the members of the Louisiana State University Staff.

The Officers of this Section for the coming year are Dr. W. Sodeman, Chairman, Dr. E. S. Kagy, Vice-Chairman, and Dr. J. Wells, Secretary.

PHYSICIANS ELECTED BY TULANE ALUMNI

Dr. Walter L. Moss of Lake Charles was selected as president of the Tulane Alumni Association when this organization held its annual meeting on Saturday, November 23. Dr. Moss graduated from the Medical School of Tulane in 1927 and when an undergraduate student was president of the Medical School student body and the University student council.

At this same meeting Dr. Ambrose Storck was elected a member of the Executive Committee and Dr. Charles Odom was selected to fill a vacancy on the Committee.

NEW HEAD OF ANATOMY DEPARTMENT AT L.S.U. MEDICAL SCHOOL

Dean Vernon Lippard has announced the appointment of Dr. Charles M. Goss as professor and head of the Department of Anatomy at Louisiana State University Medical School. Dr. Goss is well known to anatomists. At the present time he is head of the Department of Anatomy at the University of Alabama. It may be of interest to the medical profession to known that Dr. Goss is preparing a new edition of Gray's Anatomy. This standard textbook on anatomy has been published for many decades. It is probably the oldest book on any medical subject that is in active use. It has been said of Gray's Anatomy that more copies of it have been sold than any other medical book with the possible exception of Sir William Osler's Principles and Practice of Medicine.

ANNUAL MEETING OF THE FELLOWS OF THE AMERICAN COLLEGE OF SURGEONS

In accordance with Article III, Section 5, of the By-Laws, the annual meeting of the Fellows of the American College of Surgeons is called for 1:45 o'clock on the afternoon of Thursday, December 19, 1946, in the Music Hall, Public Auditorium, Cleveland, Ohio.

This meeting constitutes the annual business meeting of the Fellows of the College, at which time officers are elected and reports from officials of the College are presented. Attached is the program for the meeting.

Each Fellow of the College is respectfully urged to be present.

Irvin Abell, M.D.,
Chairman, Board of Regents

A most interesting scientific program has been arranged for this Annual Convention of the American College of Surgeons. There will be a forum on fundamental surgical problems four days in the week. There will be evening symposia in general surgery on Tuesday, Wednesday and Thursday nights and at the afternoon panel discussion which will be held Monday through Friday afternoons, there are surgical problems to be presented.

The technical exhibit, together with the registration and clinic ticket bureaus, will be held in the arena of the Cleveland Public Auditorium.

REGIONAL MEETING OF THE AMERICAN COLLEGE OF PHYSICIANS

A regional meeting of the American College of Physicians was held in Memphis on November 22, honoring Dr. Hugh Morgan, president-elect of the College. The committee in charge included governors from Texas, Arkansas, Tennessee, Mississippi and Louisiana, the latter being Dr. Edgar Hull, Professor of Medicine at Louisiana State University Medical School.

FELLOWSHIPS IN CANCER RESEARCH

Fellowships in cancer research of the American Cancer Society which is administered by the Committee on Growth of the National Research Council, are available to those who wish advanced training and experience in any field of investigation pertaining to the problems of cancer. These fellowships if on a junior basis pay from $2,000 to $3,000 a year; for the senior fellowships the stipend is from $3,000 to $6,000 per annum. The Committee on Growth reserves the right to select the institution in which the fellowship is to be applicable.

THE SOUTHERN MEDICAL ASSOCIATION MEETING

At the recent meeting of the Southern Medical Association at Miami, Florida the first part of November, Dr. Gordon McHardy was selected to be chairman of the section on gastro-enterology, Dr. Ralph V. Platou, chairman of the section on pediatrics, and Dr. Merrill C. Beck, chairman of the section on anesthesiology, succeeding Dr. John Adriani.

The following Louisiana doctors were to appear on the program either to read papers or open discussions: Drs. William A. Sodeman, Thomas Findley, Rawley Penick, Jr., Grace A. Goldsmith, Harold Cummins, Ralph V. Platou, M. T. Van Studdiford, Lee D. McLean, Arthuro Alvarado, Russell Holman, James H. Eddy, Jr., Lyon K. Loomis, Thomas Benton Sellers, W. R. Buffington, William B. Clark, George M. Haik, David A. Davis, John Adriani, Maxwell E. Lapham and John H. Whitney, all of New Orleans; C. H. Webb, S. George Wolfe, R. T. Lucas, Charles E. Anderson, Shreveport, and Fritz LaCour, Lake Charles. Drs. Grace Goldsmith, Bernard G. Efron, George Burch, Thorpe Ray, J. Brown Farrior, Neal Owens and John Adriani had scientific exhibits.

Mrs. A. A. Herold of Shreveport presided over the sessions of the Women's Auxiliary and Dr. Ruth G. Aleman was chairman of the women physicians group.

The following members of the State Medical Society were officially registered at the convention:

Drs. Maunsel B. Pearce, Monroe B. Wilson, and Allen C. Winters, of Alexandria, Drs. Guy H. Faget and F. A. Johansen, Sr., of Carville; Dr. Jos. H. Pankey of Ferriday, Dr. Pegram L. McCreary of Lake Charles, Dr. George W. Wright of Monroe, Drs. Woodward D. Beacham, Louis A. Breffeilh, Edgar Burns, Guy A. Caldwell, O. P. Daly, Jos. S. D'Antoni, Roy E. de la Houssaye, Ernest Carroll Faust, Geo. D. Feldner, John E. Garcia, C. S. Holbrook, Edgar Hull, Leo Francis Klenk, E. L. King, Edwin H. Lawson, Lucien A. LeDoux, Francis E. LeJeune, Lodwick S. Meriwether, Isidore L. Robbins, Robert. A. Robinson, Jr., Daniel H. Rowe, R. F. Sharp, Henry Ashton Thomas, H. R. Unsworth, Arthur J. Walker, and Monroe Wolf, of New Orleans, Dr. Henry S. Roans of Ruston, Dr. A. A. Herold of Shreveport, Dr. Rosamond R. Arceneaux of Welsh.

We were delighted to learn that Dr. Lucien LeDoux was elected president of the organization and that Mrs. Wiley Buffington was elected president of the Women's Auxiliary. Editorial Comment on these two elections will appear later.

OCHSNER RECEIVES TIMES PICAYUNE LOVING CUP FOR 1945

The Times Picayune announced in its November 17 edition that Dr. Alton Ochsner had been awarded the Times Picayune Loving Cup for cumulative community service. He was selected by a committe composed of representatives from the Association of Commerce, the Board of Trade and the Cotton Exchange.

The award of this loving cup to Dr. Ochsner is well merited. He has been an outstanding man in this community since he became Professor of Surgery at Tulane in 1926.

Dr. Ochsner is the second physician to receive this honor. In 1940 the loving cup was presented to Dr. Rudolph Matas.

AN IMPORTANT RESOLUTION

The following resolution was unanimously approved and passed at the Twenty-Eighth National American Legion Convention, in San Francisco, October 4, 1946.

Whereas: Veterans who have served in the armed forces now have available to them hospital and medical care provided by the United States Government; and

Whereas: There are countless voluntary health insurance plans now being offered by the physicians and the insurance companies; and

Whereas: Proposed plans of compulsory health insurance would increase the tax burden and bring about regimentation of the medical profession; and

Whereas: All forms of compulsion are repugnant to our American way of life since our liberties and opportunities would be circumscribed;

Now, Therefore, Be It Resolved: That the National Assembly of the American Legion hereby expresses its opposition to compulsory health insurance.

We are deeply indebted to Dr. Joseph C. Menendez who headed the Delegation from Louisiana to the American Legion meeting in San Francisco and to the delegates themselves for their cooperation in supporting the resolutions opposing compulsory health insurance.

Dr. Daniel Murphy, President of the Orleans Parish Medical Society, has announced approval by the Society of the plans to establish an Eye-Bank at the Eye, Ear, Nose and Throat Hospital. Approval was subject to the medical profession being in charge of all medical decisions.

This Eye-Bank will serve the Southeastern area, the eyes being transported in cooperation with the Eastern Air Lines and the Red Cross Motor Corps.

PLAUCHE HOSPITAL ASSIGNED TO OCHSNER FOUNDATION

Announcement was made approximately about the middle of November that the Plauche Hospital had been turned over to the Ochsner Foundation. Final consummation of the contract with the government will probably be completed within the next week and officially the institution will be under civilian control. The new hospital will be known as the Foundation Hospital and will be open not only to members of the Ochsner Clinic, but also to any member of the Orleans Parish Medical Society. It is expected that the painting, redecorating and rearrangement of the buildings will be completed within the next two months and that the hospital will open formally for the care of patients on February 1.

NEWS ITEMS

Dr. Mercer G. Lynch has been nominated by the Secretary of War as a Civilian Consultant to the Secretary of War in his special field of otolaryngology.

Dr. Edward L. Bortz, chairman of the Committee on National Emergency Medical Service of the American Medical Association, has announced that there will soon be mailed a comprehensive questionnaire to more than 45,000 discharged medical officers of World War II. It is urged that all medical officer veterans answer this questionnaire as promptly as possible so that it may serve as a guide in preparing for any new national emergency.

The United States Public Health Service recently dedicated the eighth building of the National Institute of Health at Bethesda, Maryland. This Memorial Laboratory will be devoted to research on the Rickettsial diseases, tularemia, psittacosis, poliomyelitis, and other central nervous virus diseases.

At Washington on November 7, twenty-four representatives met with the National Advisory Cancer Council to discuss cancer from the angle of medical education. This meeting was held under the auspices of the National Cancer Institute of Bethesda.

Representing Louisiana were Dr. M. E. Lapham of the school of medicine at Tulane and Dr. Alton Ochsner, professor of surgery at Tulane.

Over $100,000,000 has been paid by the United States Government under the Emergency Maternity and Infant Care Program. At the peak between 40 and 50,000 cases have been authorized a month. Now the authorization averages about 15,000 a month. In the state of Louisiana there were 15,200 births under the E.M.I.C. Program.

Dr. J. Brown Farrior was awarded the second prize for his scientific exhibit on the ear at the recent meeting of the Southern Medical Association.

Dr. Grace Goldsmith of the Department of Medicine at Tulane and Drs. Burch and Ray of the same department received honorable mention for their exhibits.

AMERICAN UROLOGICAL ASSOCIATION

"*Urology Award*—The American Urological Association offers an annual award 'not to exceed $500' for an essay (or essays) on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to residents in urology in recognized hospitals.

For full particulars write the Secretary, Dr. Thomas D. Moore, 899 Madison Avenue, Memphis, Tennessee. Essays must be in his hands before May 1, 1947.

The selected essay (or essays) will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Hotel Statler, Buffalo, New York, June 30-July 3, 1947".

Committee on Scientific Research
Miley B. Wesson, Chairman
Judson B. Gilbert
Anson L. Clark.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The next written examination (Part I) for all candidates will be held in various cities of the United States and Canada on Friday, February 7, 1947, at 2:00 p. m. Candidates who successfully complete the Part I examination proceed automatically to the Part II examination to be held in Pittsburgh, Pa., June 1-7, 1947. All candidates, especially those in military service, are requested to keep the Secretary's Office closely informed of changes in address.

Several changes in Board regulations and requirements were put into effect at the last annual meeting of the Board held in Chicago, Illinois, from May 5 to May 11, 1946.

For further information and application blanks address—Paul Titus, M.D., Secretary, 1015 Highland Building, Pittsburgh, 6, Pennsylvania.

MISSISSIPPI STATE MEDICAL EDUCATION PROGRAM

The Mississippi State Medical Education Board announces the granting of 47 scholarship loans effective the first semester of 1946-47. These loans have been awarded to young men and women from 28 towns in 23 counties in Mississippi. They will attend different medical schools, many of them taking their preclincal work at the University of Mississippi Medical School, a two-year institution. The students in the clinical branches will attend 11 different medical schools throughout the nation. The majority of these will go to Tulane University, with Louisiana State University and the University of Tennessee Colleges of Medicine having an equal number.

The action taken by the Mississippi legislature in granting the scholarship loans to prospective medical students and doctors is, we believe, one of the most outstanding advances in the medical education that has been consummated in recent years. It is to be borne in mind that medical schools can only be established satisfactorily in large centers of population and there are very few states in this country that have sufficiently large hospitals for the adequate training of medical students. It would be very much better for many more were schools to follow the Mississippi plan than it would be to attempt to carry on with a lack of clinical material and with a faculty which will show marked weaknesses in certain divisions. Notably those are the clinic divisions because there are not enough well-trained physicians in the locality who are interested in training students and devoting time to the teaching hours that are required.

FELLOWSHIPS IN MEDICAL RESEARCH

The Surgeon-General of the United States Public Health Service, Dr. Thomas Parran, has announced that approximately 120 one-year fellowships in medical research are open to graduate science students. The details of these research fellowships may be obtained from Dr. Dyer, Director of the National Institute of Health, Bethesda, Maryland.

NOVEMBER ISSUE WANTED

Doctors who do not wish to retain copies of the New Orleans Medical and Surgical Journal are requested to return copies of the November, 1946 issue to the office of the Journal due to a shortage of extra copies of this issue.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending October 5 there were 141 deaths in the city of New Orleans, divided 93 white and 48 colored. Of these deaths 21 were children under one year of age, 15 white and six colored. These figures were considerably higher than for the last week reported which ended September 28 in which there were 122 deaths. The following week, which closed October 12, there was a marked diminution in the number of deaths that took place in the city. In this week there were 72 white people who died, 35 colored, with 15 of the deaths occurring in children under one year of age. Strange to say, 13 of these deaths were in white children which is quite unusual. For the following week, terminating October 19, the death rate jumped materially because of the death of 142 of the citizenry of the city, of whom 95 were white, 47 were colored, with only 11 deaths in children under one year. There seems to be rather a marked fluctuation in the number of death in the city from week to week; for that which closed October 26, there was a considerable fall with 119 deaths reported, 78 of which were white and 41 were colored, with 13 children dying this week, eight of whom were colored. The week which came to an end on November 2 presented figures almost identical with the previous week. There was one fewer deaths than the week before. Of the 118 people who died in the city, 75 were white, 43 were colored and ten were children under one year old.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending October 5, showed the following reportable diseases in numbers greater than ten; cancer 173, pulmonary tuberculosis 122, unclassified pneumonia 111, hookworm infestation 14 and poliomyelitis 11. The poliomyelitis cases were reported throughout the state with no one parish having more than three cases originating within its boundaries. There was a rather marked difference from the previous week the following week in the number of reported cases, there being only three occurring in numbers greater than ten, these being pulmonary tuberculosis 63 cases, hookworm infestation 21 and typhus fever 16. There were nine cases of poliomyelitis reported this week, again the cases being scattered throughout the state. Of the large number of typhus fever cases, 16 in all, eight of them were reported from Orleans Parish. For the week which closed October 19, pulmonary tuberculosis again led all reportable diseases with 43 cases, followed by 37 of malaria, 29 of influenza, 20 of septic sore throat, and 14 of hookworm disease. Thirty-five cases of malaria were reported from Jackson Parish. Seven cases of poliomyelitis were reported this week, three of which came from Tangipahoa Parish and two from Orleans. The week which ended October 26 was an unusually healthy week, there being reported only three diseases in numbers over ten, these being hookworm infestation 25, pulmonary tuberculosis 21 and scarlet fever 13. The seven cases of poliomyelitis that were listed developed in six different parishes widely scattered over the state.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending September 30, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	49	578	527
Gonorrhea	1246	13257	14503
Granuloma Inguinale	22	219	241
Lymphopathia Venereum	8	83	91
Syphilis	1580	13760	15340

NAVAL AIR RESERVE TRAINING COMMAND
U. S. NAVAL AIR STATION
GLENVIEW, ILLINOIS

Secretary Louisiana State Medical Society
1430 Tulane Avenue
New Orleans, Louisiana

Dear Doctor:

Attached herewith is a rewrite of the need of Naval Medical Officers in the Naval Air Reserve Training program.

Circulation of the contents of this notice among the members of your State organization will be sincerely appreciated as it is believed that many Ex-Naval Medical Officers would be desirous of continuing their association with the Navy on a full time basis under conditions outlined or to a limited extent with the Naval and Marine air groups of the Naval Reserve in regular periods of training duty.

Thanking you in advance for your cooperation.

Yours very truly,
G. B. Ribble,
Captain (MC) USN
Staff Medical Officer

ATTENTION NAVAL RESERVE OFFICERS!

Full Time Active Duty: The attention of Reserve Medical Officers and of Pharmacists of the Hospital Corps is invited to the opportunity to perform *full-time active duty* at one of the major naval air stations of the Naval Air Reserve Training Command or at one of the Naval Air Reserve Training Units (NARTUs) listed below.

Stations of the NARTC

NAS, Atlanta, Ga.
NAS, Columbus, Ohio.
NAS, Dallas, Texas.
NAS, Glenview, Ill.
NAS, Grosse Ile, Mich.
NAS, Los Alamitos, Cal.
NAS, Memphis, Tenn.
NAS, Minneapolis, Minn.
NAS, New Orleans, La.
NAS, New York, N. Y.
NAS, Oakland, Cal.
NAS, Olathe, Kan.
NAS, Squantum, Mass.
NAS, St. Louis, Mo.
NAS, Willow Grove, Pa.

NARTUs of the NARTC (Based at the below listed Naval Air Stations)

Anacostia, D. C.
Jacksonville, Fla.
Miami, Fla.
Norfolk, Va.
San Diego, Cal.
Seattle, Wash.

How to Apply: Officers in the above category who are interested in *full-time active duty* as a member of the stationkeeper staff at one of the stations or units listed above should initiate letters to the Bureau of Naval Personnel, via the Chief of Naval Air Reserve Training, Naval Air Station, Glenview, Ill., and BuMed, listing three or four stations at which duty is desired in order of preference. Personnel are desired in the ranks of Commander and Lieutenant Commander in the Medical Corps.

Advantages: Officers qualifying for the above billets can be assured of every consideration in obtaining and continuing duty at the station of their preference. It is the policy of the Command to obtain "orders to duty involving flying" for designated Naval Flight Surgeons. Government quarters are available at many of the major Naval Air Stations.

Part Time Duty: Naval Flight Surgeons of the Reserve who are desirous of affiliating themselves with either the Organized or Volunteer components of the Inactive Reserve composed of Naval or Marine air groups training at one of the Naval Air Stations or NARTUs listed above should contact the Commanding Officer of the station or the NARTU at which the training unit is based.

Do not miss this splendid opportunity to enjoy an indefinite tour of "shore duty only" in a peace time Navy at one of the Naval Air Stations listed above, thereby both benefiting yourself and the Navy by contributing your services to the post-war Naval Air Training Program!!

BROOX CLEVELAND GARRETT, M.D.
1888-1946

Dr. B. C. Garrett, of Shreveport, died on November 15, 1946, after a protracted illness. In the passing of Dr. Garrett many medical men throughout the state will lose a good and loyal friend. He was universally popular and very generally liked by every one.

Dr. B. C., as he was generally known, graduated from Tulane in 1913 and two years later became a member of the State Medical Society, holding active membership until elected an honorary member in 1946. He was a college athlete of renown and all his life his chief avocation with connected with outdoor activities; hunting, shooting and farming, but to Dr. Garrett there was not much time for play as after entering practice he rapidly developed one of the largest surgical practices in

Shreveport which he maintained up until the time of his last illness. In the death of Dr. Garrett a great many of us have lost a real and true friend, a man who could be depended upon in times of difficulty and who was always willing to help not only his friends but all others.

LESLIE LEON LUMSDEN
1875-1946

One of the outstanding leaders in the development of public health in the United States, Dr. Leslie L. Lumsden, died November 8 at the United States Marine Hospital, New Orleans. His death at the age of 71 was due to leukemia complicated by pneumonia.

From the time that Dr. Lumsden was commissioned an Assistant Surgeon in 1898 until he retired in 1939, he consistently shattered the accepted beliefs of his profession. By tireless research and field investigations, Dr. Lumsden exposed true vectors of disease, definitely established sources of contamination in the community, and originated and developed full time rural health plans.

He uncovered all the facts concerning the spread of typhoid fever by means of unsanitary food and water as well as flies and other carriers. The reports of his investigations served as the basis of future control measures for the disease.

BOOK REVIEWS

Early Ambulation and Relative Procedures in Surgical Management: By Daniel J. Leithauser, M. D., F. A. C. S. Springfield, Illinois, Charles C. Thomas, 1946. Pp. 232. Price, $4.50.

This volume excites interest and emotion without necessarily stimulating enthusiasm.

The easy complacency of the author in regard to his own attitudes and his assumption that the opinions contrary to his own are based on ignorance and tradition, is to say the least interesting.

This small volume of two hundred odd pages is a compendium of the author's interpretation of physiology, anatomy, surgical technic, surgical nursing, and testimonials from patients.

I fear that the author has not given just credit to surgeons whose procedures for preventing postoperative complications and disabilities differ from his own. In all matters controversial, time and judgment of the majority will provide an adequate answer.

ISIDORE COHN, M. D.

Jewish Luminaries in Medical History: By Harry Friedenwald, M. D., D. H. L. (Hon.), D. Sc. (Hon.). Baltimore, The Johns Hopkins Press. 1946. Pp. 199. Price, $3.00.

This volume has many distinguishing characteristics. It is a choice morsel from the pen of the greatest living student of that phase of medical history which has to do with the part played by Jews in the development of medical practice.

Dr. Friedenwald's monumental, epoch making, two volume work on "The Jews and Medicine" was published in 1944 by The Johns Hopkins Press.

Dr. Friedenwald gives due credit for his interest in medical history to Osler, Welsh, and his own father, the late Dr. Aaron Friedenwald. By this tribute Friedenwald glorifies the work of his preceptors, and by his work his name is added to the list of great medical historians.

Sigerist states in the foreword that Friedenwald is a great book collector. He adds "Unlike other collectors, he reads his books, studies them, and uses them as historical sources."

As a witness to the truth of this statement, one has but to turn to the catalog (which forms part of this volume)—"Of works bearing on the subject of the Jews and Medicine from the private library of Harry Friedenwald," to realize that in his library may be found the finest collection on the subject in the United States, and, I would venture, probably the equal of any in the world.

Students of medical history will find a library incomplete without the two volume work of Friedenwald's on "The Jew in Medicine" and this essay on "Jewish Luminaries in Medical History" along with the catalog of Friedenwald's library.

ISIDORE COHN, M. D.

Pneumoperitoneum Treatment: By Andrew Ladislaus Banyai, M. D., F. A. C. P., F. C. C. P. St. Louis, C. V. Mosby Co., 1946. Illus. Pp. 376.

An authoritative monograph on the pneumoperitoneum treatment of pulmonary tuberculosis is quite timely in view of the present widespread interest in this method. The author of this book, both by priority and by sustained interest in the subject, is probably the best qualified man in this country to do it full justice. He has covered it exhaustively from the introductory historical review to the bibliography of almost 700 references (including 24 of his own papers on pneumoperitoneum). Everything possibly related to pneumoperitoneum is there. Therefore the result is a reference work rather than a practical manual to guide the physician who wishes to employ this form of treatment. The 32 pages devoted to technic of administration will be found useful as will also the 130 pages on the treatment of pulmonary tuberculosis. Some question might be raised as to the validity of the physiologic concepts in the discussion of intra-abdominal pressure. This method in the treatment of all the conditions covered in the other ten chapters on therapeutic application is at best of doubtful utility. The illustrations are numerous and adequate to show the gross roentgenologic features of the method although finer details are lost in the reproduction. This monograph may profitably be referred to by anyone interested in the treatment of tuberculosis or other pulmonary diseases. However it should be read with the realization that it is not a critical analysis of a method of treatment which is still under trial and must always be supplementary to more basic forms of therapy.

J. L. WILSON, M. D.

Peptic Ulcer, its Diagnosis and Treatment: By I. W. Held, M. D., F. A. C. P. and A. Allen Goldbloom, M. D., F. A. C. P. Springfield, Ill., Chas. C. Thomas, 1946. Pp. 382. Price, $6.50.

This small concise volume of three hundred and eighty-two pages lives up to the authors' claim that it is a clinical treatise on the diagnosis and treatment of peptic ulcer. The contention that it is suited for the practitioner is probably true; the reviewer was not impressed with its applicability to the physician in the military service; the student can get as much information from the average endorsed text book on internal medicine. It is apparent that I am not impressed that this is an authoritative treatise on peptic ulcer.

In the section on pathogenesis the authors indifferently enumerate the possibilities, briefly treat on the all important neurogenic factors and completely omit any reference to the experimental production of peptic ulcer by histamine.

The chapter on pathology is not impressive.

In consideration of symptomatology proper emphasis is given to the periodicity of pain.

A relatively too elaborate consideration is given the differential diagnosis of a rather clear cut clinical syndrome applicable to a perforated ulcer.

The good point on the consideration of hemorrhage is that persistent severe bleeding probably indicates pancreatic duodenal vessel erosion.

In the management of stenosis the authors are extremely vague as to where the internist gives up to the surgeon. Again the same vagueness exists as to when to elect surgery in a gastric ulcer not responding to medical measures. Like so many other authors here again too much emphasis is placed on the site of an ulcer as to its malignant potentialities when every good clinician knows carcinoma can occur in any portion of the stomach.

The indications for surgery are well outlined. The types of operative procedure are handled too indifferently even for an internist. No mention is made of vagotomy nor of jejunal transplants.

The therapeusis is poorly and indifferently presented with the opening statement that there is "no specific treatment". One can not manage ulcer patients well with such an attitude, there may be no specific treatment but there is an acceptable method of management of disease and patient. The mention of such discarded products as Larostidin and Synodal without giving an evaluation of their efficacy is not commendable.

The reviewer is interested to know there is a "flexible esophagoscope".

The entitlement Gastric Catarrh is meant to indicate the established entity gastritis. The chapter is extremely poor, does not approach a comprehensive knowledge of the subject and omits the essential feature to the entity, i.e., the gastroscopic evaluation.

A chapter dealing with the "dyspeptic soldier" insults him by failing to conclude the possible organic background for his symptomatology, without admitting the inadequacies of many military examinations and follow-up studies.

The portion dedicated to roentgen evaluation seems adequate. It is surprisingly well done when one considers this in contrast to the mention of the existence of gastroscopy and the absence of mention of gastrophotoscopy. One finds figures No. 27 and No. 33 reversed.

I wonder if many clinicians consider that the manifestations of a peptic ulcer call into consideration for differential diagnosis such entities as: mucus colitis and dibothrophillin latum infestation; the authors do.

While interesting themselves too surgically in the immediate postoperative management the authors neglect to consider the many physiologic disturbances that occur as to result of surgery.

I am not inclined to endorse this volume as an authoritative treatise on the diagnosis and treatment of peptic ulcer. It is well arranged, easily readable and is adequately illustrated.

GORDON MCHARDY, M. D.

PUBLICATIONS RECEIVED

The Dial Press, New York: The Challenge of Polio, by Roland H. Berg.

Grune & Stratton, New York: Clinical Methods of Neuro-Ophthalmologic Examination, by Alfred Kestenbaum, M. D.

Mead Johnson & Company, Evansville, Indiana: Courage and Devotion Beyond the Call of Duty. A Manual for the General Practitioner.

Henry Schuman, New York: Memoir on Sulphuric Ether, by W. T. G. Morton. Victory Over Pain, by Victor Robinson, M. D. The Centennial of Surgical Anesthesia by John F. Fulton, M. D., and Madeline E. Stanton, A. B. Journal of the History of Medicine and Allied Sciences, a Quarterly.

Commonwealth Fund, New York: Dentistry, An Agency of Health Service, by Malcolm Wallace Carr, D. D. S.

New Orleans Medical
and
Surgical Journal

Vol. 99 JANUARY, 1947 No. 7

MODERN SURGICAL PROCEDURES FOR LESIONS OF THE COLON AND RECTUM*

HOWARD MAHORNER, M.D.
AND
JOSEPH SABATIER, M.D.†
NEW ORLEANS

In the not too distant past surgical removal of a lesion of the colon or rectum was accomplished routinely by multiple procedures. This necessitated prolonged hospitalization. The control of a group of factors newly appreciated during the last decade have permitted a change in this attitude so that modern operations for colon and rectal lesions usually involve a very short hospital stay and a short period of morbidity. Furthermore, the risk of operation has been reduced even though stage procedures now may largely be eliminated. Formerly preliminary colostomy was a procedure of choice prior to rectal resections; and preliminary cecostomies, or so-called defunctioning colostomies, were considered proper prior to removal of other lesions of the large bowel. Now these stages of operations are almost entirely obsolete.

Among the factors which have changed advanced surgical thought in this field are the following. First, altered fluid and chemical balance of the blood. Measures may be taken in the preoperative preparation to obviate variations from the normal. It is now known that frequently patients with carcinoma have dehydration and altered blood chemistry, low blood protein, avitaminosis, diminished erythrocyte count. These features which influence the risk of operation may be corrected to a relative normal in the preoperative phase by administration of transfusions, amino acids and vitamins. Secondly, it is absolutely essential that the bowel be prepared. Optimum preparation of the large bowel for operation means highest obtainable emptyness. To attain this it is essential that the patient be fed a non-residue diet for a period of at least 48 hours immediately preceding operation. This tends to create further protein deficiency which must be combatted by parenteral administration of protein. It is a mistake to feed a patient right up to the time of operation with the intended motive of retaining normal blood proteins. What is gained in chemical balance is lost by the insecurity of not having an empty colon at the time of operation. It is much more important to have the bowel empty and for this reason it is imperative that replacement of the lost protein due to diminished diet in this immediate preoperative phase, be made by the intravenous route.

Chemotherapy is another important advance. One has the choice of administering poorly absorbed sulfonamide (succinylsulfathiazole, sulfathalidine) with the hope that it will diminish the bacterial content of the colon and thus reduce the risk for operation. In the past we have employed these drugs in a desultory manner with no real conviction of their efficacy. Finally it has been discarded from our routine with the belief that it is inconsequential in the preparation of the patient, and we shall not revert to its use until more convincing evidence of its value is published in the literature.

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*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

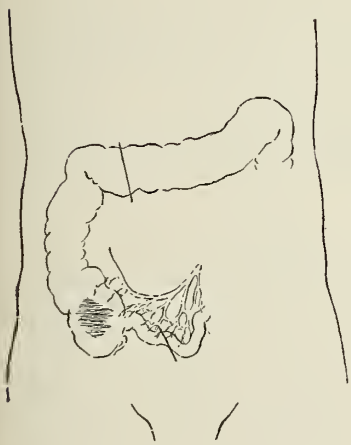
†From the surgical services of Dr. Howard Mahorner, Baptist Hospital and Touro Infirmary, New Orleans, La.

Other types of chemotherapy have played a tremendous part in reducing mortality for these operations. Penicillin, particularly, protects the patient against contamination and peritonitis. It is our routine to start the administration of penicillin the night before the operation. Following the operation penicillin is administered intramuscularly and sulfadiazine intravenously.

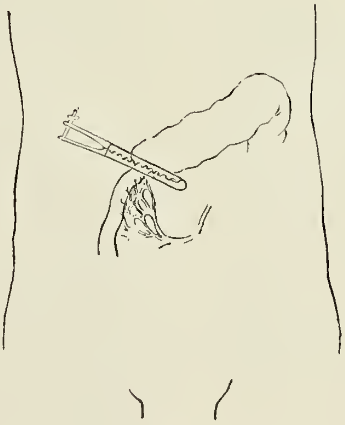
Other important factors in the success of this type of surgery involve the support of the patient's general condition during the operation by administration of whole blood. Equally important is the selection of an anesthetic which will not add to the patient's burden and which will permit the surgeon to work with greatest ease and with least trauma to the patient. Spinal anesthesia is the optimum anesthetic for this type of surgery.

The diagnosis of carcinoma of the colon and rectum is so important that it would not be proper in a paper on operative procedures for lesions of these organs, to omit entirely some word regarding important features in the diagnosis. The symptoms and objective findings of carcinoma of the large bowel and rectum may be separated into three main groups depending on where the lesion is located. Carcinomas of the cecum and the first portion of the ascending colon rarely produce complete obstruction. They may subjectively result in crampy pain, but weight loss, anemia and objective evidence of a mass, together with the roentgenographic evidence of a lesion are the important features in establishing a diagnosis. Lesions elsewhere in the colon down to the rectum proper are apt to produce obstructive symptoms—and bleeding. With or without objective findings of a mass and distension, but with proof by retrograde barium enema of the existence of a filling defect, the diagnosis may be established. Lesions of the sigmoid and descending colon may bleed as an initial symptom but they frequently produce obstruction without other signs. An early carcinoma of the rectum never obstructs. Small carcinomas of the recto-sigmoid obstruct but carcinomas of the rectum must be huge to produce obstruction. The ampulla of the rectum is so large it will accommodate a considerable growth without compromising the lumen to the extent of obstructing. The patient does not have obstructive symptoms, he does not have constipation unless the growth is huge and the symptomatology unduly prolonged. Patients have, early, the sensation of incomplete evacuation of the bowel and that there is something there that cannot pass. Bleeding is the most common, single symptom of carcinoma of the rectum. Eighty per cent of carcinomas of the recto-sigmoid and rectum cannot be touched by the finger on digital examination. If the growth is in the ampulla between the recto-sigmoid and the lower end of the rectum, the finger may gain the sensation that there is a mass even though the lesion may not be actually touched by the finger. It is well worth knowing that a carcinoma of the rectum is not disclosed by a barium enema. Carcinomas of the recto-sigmoid and sigmoid and the remainder of the colon may be disclosed by a barium enema, but the rectum itself is such a large space that a filling defect from a small growth on the posterior or interior surfaces is usually overlooked by roentgenologists. Diagnosis of carcinoma of the rectum must be made by proctoscopic visualization and by biopsy of the lesion. The fact that there is a segment of the rectum whose diagnostic domain is that of the proctoscope only, is not sufficiently appreciated.

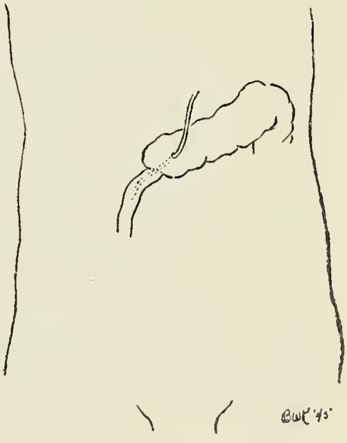
Review of our operations on the colon and rectum for the years 1943 through April, 1946, inclusive, showed 33 resections of the colon and rectum. Thirty-one of these procedures were primary resections without preliminary colostomies. Two resections were done after preliminary colostomies. One of these preliminary colostomies was made because of obstruction by a lesion of a descending colon. The other preliminary colostomy was an elective one, preceding an abdomino-perineal resection of the rectum. This was done in January, 1943, and is the first case in this series, the last case in which an elective preliminary colostomy was performed. Thirty of these procedures were done for malignancies; three were done for benign lesions; one granuloma of the cecum, two obstructive lesions of the transverse colon resulting in segmental megalocolon proximal to the obstruction. Six right hemicolectomies were done and four segmental resections of the transverse, descending colon and sigmoid. All of these were primary resections with immediate end to end anastomoses. All of these anastomoses were made aseptically usually by employing a Furniss clamp. Even in resection of the terminal ileum and right hemicolon an end to end anastomosis was made. Under these circumstances Witzell type enterostomy was employed. The catheter was inserted in the colon 6 centimeters distal to the anastomosis and the enterostomy catheter was passed cephalad into the proximal segment (figure 1). This insured decompression of the proximal segment just above the suture line and was proof positive that the anastomosis was patent. In doing segmental resections of the colon an enterostomy is made with this arrangement of the enterostomy catheter. This catheter removed on the tenth or twelfth postoperative day may be used prior to that for installation of oils to facilitate passage of intestinal contents through the anastomosis.



1. Lesion and area resected



2. Anastomosis with Furniss clamp



3. Catheter enterostomy retrograde and transanastomotic

There were 23 resections of the rectosigmoid and rectum in this group. In 10 instances primary anterior resection of the rectosigmoid or the rectum was done with anastomosis of the sigmoid to the rectal stump. In addition to that in five instances in which the growth was too low to reestablish continuity after resecting the rectum anteriorly an abdominoischeal resection previously described by the senior author[1] was performed. In this operation after mobilizing the rectum anteriorly and dropping it into the pelvis as in an abdomino-perineal resection, it is removed through an incision in the ischeal fossa and the sigmoid is brought down and anastomosed to the stump of the rectum just above the anal canal. It is questionable whether this operation is going to be justifiable. Growths this low are so near the sphincter that preservation of the sphincter may not be feasible without increasing the danger of recurrence.

In eight instances abdomino-perineal resections were done. This group contains the earlier cases of those reported herein and undoubtedly a few of those would be done today by an anterior resection with reanastomosis.

There were three deaths in the entire series, one a woman age 76 who died on the table following resection of the cecum and ascending colon and anastomosis. This we felt was an anesthetic death since there was a sudden irreversable apnea when the spinal anesthesia was supplemented by general inhalation anesthesia at the termination of the operation. One patient, aged 32 years, following resection of the rectum with re-anastomosis, died on the fifth postoperative day. The findings were generalized peritonitis. The third death was due to hemorrhage following abdomino-perineal resection in an extremely obese female. In the group of 15 patients in whom continuity was re-established after resection of the rectum there was one death mentioned above.

There is a great debate in the literature on whether conservative operation should be done for cancer of the recto-sigmoid and rectum. In the operation described by Dixon the upper segment is removed just as radically as is the removal of this same segment in abdomino-perineal resection. Indeed that part of the operation is identical with abdomino-perineal resection. In those instances, however, in which the continuity is re-established the distal part of the procedure is less radical. Numerous studies are being shown including those of Gilchrist and David;[2] and Coller, Kay and McIntyre;[3] and Waugh[4] which demonstrated significant distal spread of cancer of the recto-sigmoid and rectum in only 2 to 3 per cent of all resected specimens. In the group in which distal metastases were found lymph nodes proximal to the lesion were so obstructed that it became apparent that the cause of the distal metastases is due to retrograde flow of lymph induced by obstruction of nodes in the normal direction of flow.

The risk of operation for anterior resection and anastomosis is probably about the same as abdomino-perineal resection. For both operations it should be extremely low in a sufficiently large number of patients. Recurrence rate has yet to be proved. Only the series of Dixon[5] and of Babcock and Bacon[6] are sufficiently large to have a definite value for comparison of three years' cures between the results of abdomino-perineal resections and more limited resections with preservation of the sphincter. The results published so far indicate that the recurrence rate is just as low after preservation of the sphincter in those lesions which are above the distal 8 to 10 centimeters of the rectum, as it is following abdomino-perineal resection for a similar group of cases.

Numerical considerations are not the only ones. The number of lives saved at the time of operation will be one of the definite determinants for the choice of operation; but other things are to be considered. People who are left with a permanent colostomy are not restored to normal life. Those in whom the continuity is restored by an anterior resection have good function of their sphincters and are restored to normal bowel function. It is not likely that for the higher lesions of the rectum and recto-sigmoid there would be any greater recurrence from the one operation or the other. But if there were a slightly greater chance of recurrence following restitution of normal continuity it may be justified on the grounds that many of the patients who are saved with colostomies are broken in spirit and morale to such an extent that in reality they would be more fortunate to have lost at the time of their operation. Forty people saved without a colostomy may be worth 50 people saved with permanent colostomy.

SUMMARY

We reported a group of 33 resections of the colon and rectum, 31 of which were done in one stage without preliminary colostomy. There were three deaths, one following resection of the ascending colon, one following abdomino-perineal resection. Attempts to reestablish continuity were made in 15 of the 23 cases of cancer of the recto-sigmoid and rectum. One only of these patients died.

We prefer single stage operations for lesions of the colon and rectum and restitution of normal continuity following resection of the recto-sigmoid and rectum for lesions at least 8 centimeters above the ano-cutaneous line. This will comprise 80 per cent of the cancers of the recto-sigmoid and rectum.

LEGEND FOR FIGURE ON PAGE 315

Fig. 1. Type of primary resection for carcinoma of the cecum or ascending colon. Following right hemicolectomy an end to end aseptic anastomosis is made. The ileum is cut obliquely to correct its adaptation to the colon. A catheter enterostomy is made with the catheter entering the colon and passing through the anastomosis into the proximal segment. The catheter is removed in about ten days.

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TREATMENT OF CHOLEDOCHOLITHIASIS WITH INTRADUCTAL INJECTIONS OF ETHER

CASE REPORTS

MARSHALL L. MICHEL, M. D.†
NEW ORLEANS

The technical difficulty involved in dislodging common duct calculi is well known to all surgeons who are familiar with biliary operative procedures. The experience of being unable to dislodge a common duct stone or of overlooking one (or more) when performing choledochotomy is not uncommon and is an admitted experience of a large number of reputable abdominal surgeons.

There are many contributing factors which often cause choledochotomy to tax the skill of the best surgical technicians. The patients are often markedly adipose. Extensive inflammatory changes (exudate, edema, adhesions, and so on) are often present in the region of the gastro-hepatic omentum. Anomalies of the ducts and blood vessels are relatively common. The patient's general condition may sometimes be such that prolonged operative procedures are contraindicated. Occasionally, common duct calculi are situated in diverticuli of the wall of the duct and choledocholithotomy is extremely difficult.

In some cases, mobilization of the duodenum to expose the retroduodenal portion of the choledochus as well as the procedure of transduodenal exploration of the terminal portion of the common duct may even prove unsuccessful.

It is, therefore, logical that the use of various chemicals in the form of common duct irrigation partially to dissolve the stones and cause them to be extruded into the duodenum has been conceived and successfully used on numerous occasions.

Any substance so employed must fulfill two important requisites: (1) the substance must have a solvent action on the stone; (2) it must also be innocuous to living tissues.

Various chemicals have been used for common duct irrigation. Ether,[6] turpentine,[7] ether-glycerine mixture,[6] ether-alcohol mixture,[6] and recently chloroform,[3] have all been used with varying success. Ether has been more widely used than any of the other mentioned chemicals and is generally accepted to be the most ideal substance for irrigation of the choledochus to remove calculi.

Pribram,[4] Walters and Wesson,[6] Burgess,[2] Boyce[1] and others have agreed on the safety and efficiency of ether in the dissolution of common duct calculi.

The solubility of cholesterol, bile pigments and bacterial and cellular debris (all components of gall stones) in ether is an accepted chemical fact. Although the degree of solubility is slight, it is sufficient to diminish the size of the calculi to such an extent that they will eventually pass into the duodenum.

Raffi,[5] has shown experimentally in dogs that repeated ether injections are relatively harmless to the common duct mucosa. These injections were carried out over much longer periods of time than are used in the treatment of actual human patients.

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Recently, Narat and Cipolla[3] have advocated common duct irrigation with warm chloroform for the dissolution of stones. It is indisputable that chloroform is a more potent solvent for biliary calculi. However, chloroform is a well known hepato-toxic agent and more careful animal experiments are necessary before chloroform can be injected into the human choledochus with any degree of impunity.

TECHNIC

If postoperative cholangiography reveals the presence of a residual common duct stone, injection of the duct with ether should not be undertaken until the following four conditions are fulfilled: (1) The patient is afebrile; (2) the operative wound is well healed; (3) icterus index is within normal limits; (4) liver function has returned to or remains within normal limits (as indicated by the cephalin flocculation and other liver function tests).

The T-tube and common bile duct should first be irrigated with normal saline and then aspirated. The patient is given a hypodermic injection of amyl nitrite. Immediately following this, 3 to 5 c. c. of ether are injected into the T-tube and the tube is pinched off until the patient complains of severe pain. At this time, the tube is immediately released and the excess ether is allowed to run back out of and evaporate out of the open end of the T-tube.

This procedure should be performed twice daily for a period of five to seven days. After this time, cholangiography should again be performed to see if the stone has passed into the duodenum.

If the above technic has been carried out and after five to seven days the stone is still present in the common duct, it is highly improbable that further irrigations will be successfull and secondary choledochotomy is indicated.

CASE NO. 1

Mrs. T. C., a white female, aged 51, had indigestion and upper abdominal pain for three years. These symptoms were particularly aggravated by the eating of fatty foods.

For three weeks prior to admission, she had severe pain in the right upper quadrant and jaundice. Typical biliary colic had occurred on several occasions. The stools were clay colored. She had fever up to 103° and had experienced severe chills.

Physical examination revealed marked tenderness in the upper right quadrant, but there was no palpable mass. The icterus index was 120. A gastrointestinal series was negative for duodenal or pancreatic pathology.

At operation, cholecystectomy and choledochotomy were performed. The common duct was explored both transduodenally and from above. Six small calculi of mixed variety were removed from the choledochus. The duct was probed without difficulty into the duodenum and the sphincter of Oddi was dilated. A T-tube was left in place in the common duct.

On the tenth postoperative day, cholangiography revealed the presence of a stone at the ampulla. The duct was incompletely obstructed and the stools were normal in color (Fig. 1).

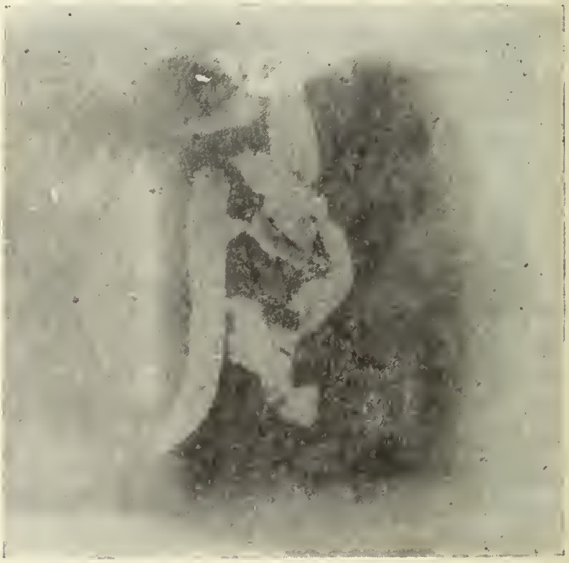

Fig. 1. Cholangiogram showing calculus in region of ampulla remaining after choledochotomy.

Ether injections were begun and carried out daily for five days. The above technic was used. Cholangiography was performed subsequent to the ether irrigations. The stone was found to be no longer present and the duct was unobstructed (Fig. 2).

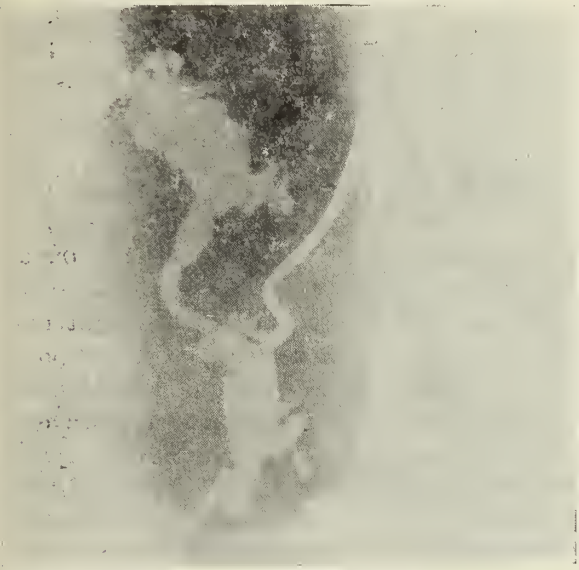

Fig. 2. Cholangiogram following ether irrigations showing calculus no longer present.

The patient at the present time is in excellent health and has had no recurrence of biliary symptoms.

CASE NO. 2

Mrs. S. L., a white female, aged 40, had had recurrent attacks of right upper quadrant pain, nausea and vomiting for the past two years. Indigestion was caused by fatty foods. Several severe attacks of pain and jaundice had occurred. The jaundice had subsided spontaneously on each occasion, but had been persistent after the most recent attack. The patient had had no chills and at no time had experienced high fever.

Physical examination revealed tenderness in the epigastrium and upper right quadrant. The icterus index was 40. The gastrointestinal series was negative for duodenal or pancreatic pathology.

After preoperative preparation, operation was performed. Cholecystectomy and choledochotomy were performed. Two small calculi were removed from the common bile duct. The duct was irrigated, the sphincter of Oddi was dilated, and a probe could be passed without difficulty into the duodenum.

On the eight postoperative day, cholangiography was performed. It was found that a defect was present in the ampulla, causing incomplete obstruction of the common bile duct. This shadow was characteristic of that caused by a calculus.

Ether irrigations, carried out daily for four days according to the above technic, were again successful in dislodging the stone.

The patient is well at the present time and has had no recurrence.

COMMENT

Two cases of multiple common duct calculi are herein reported. In each case, it was believed that adequate choledochotomy had been performed. Subsequent cholangiography revealed the presence, in each case, of a stone lodged at the ampulla, which had been missed at operation. In both instances, the stones were causing only incomplete obstruction of the duct and might have been overlooked if cholangiography had not been performed.

Ether irrigations, according to the technic described herein, were successful in dislodging the calculi.

It is to be noted that such calculi causing incomplete obstruction may be entirely asymptomatic and the stools may be normal in color. However, recurrent complete obstruction of the choledochus is inevitable in such cases. Therefore, cholangiography should always be performed after choledochotomy, regardless of the normal appearance of the stool, the absence of symptoms, and the diminution of the degree of jaundice.

Furthermore, in such cases of choledochus calculi producing incomplete obstruction, if the T-tube is removed, the remaining biliary fistula may close. Thus, the surgeon may have a false sense of security and believe that the common duct is entirely free of calculi.

The exact *modus operandi* by which ether irrigations are successful in removing common duct calculi is worthy of discussion. The solubility of the various components of biliary calculi in ether is not sufficient entirely to explain the success of ether irrigations in such cases. It is entirely possible that the marked irritation of the ether and the increased intrabiliary pressure produced by evaporation of the ether cause a propulsive reflex action in the common duct which may have something to do with the calculi being expelled into the duodenum.

SUMMARY

1. The removal of common duct calculi requires the highest degree of operative skill.

2. Common duct calculi are frequently

overlooked when choledochotomy is performed.

3. Cholangiography should always be performed before a T-tube is removed from the choledochus.

4. Numerous chemical substances have been advocated for common duct irrigation in order to remove calculi.

5. Ether, experimentally and clinically, has been reported to be safe in the removal of common duct calculi.

6. Two cases are herein reported in which common duct calculi were overlooked during the procedure of choledochotomy and cholecystectomy.

7. In each instance, the calculi were successfully removed by irrigations of the duct with ether.

8. The importance of performing cholangiography after choledochotomy is stressed.

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ESSENTIAL FACTORS IN PREOPERATIVE, OPERATIVE, AND POSTOPERATIVE CARE OF GYNECOLOGICAL PATIENTS[*]

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When the gynecological patient is to undergo an operative procedure the physician in charge should be certain nothing is overlooked that might compromise the patient's chances for a successful outcome.

It has been said that a generation ago surgery was being made safe for the patient; today efforts are being made to make the patient safe for surgery. The progress made in surgery during the past generation has been mainly in the field of pre and postoperative care.

In observing the care of patients on a large gynecologic service at the Charity Hospital in New Orleans, the thought occurred that it might be of interest and possibly of some value to review in a general way and to emphasize certain factors essential to the care of these patients before, during, and after surgery. The present low mortality and low incidence of complications postoperatively observed at this institution are due to the adherence to certain criteria based on sound surgical principles.

POSTOPERATIVE CARE

The late John Polak,[1] in a paper entitled "What Can We Learn From a Study of Mortalities?" observed that perhaps three-fifths of the deaths following his own elective gynecologic surgery might have been prevented by stricter adherence to the criteria which he had set forth. The late Hilliard E. Miller,[2] in an analysis of 154 deaths associated with benign gynecologic disease, stated that 73 of these were possibly preventable had such avoidable errors as; (1) inadequate investigation; (2) poor preoperative preparation; (3) poor or delayed or inadequate postoperative therapy; (4) lack of alertness in detecting postoperative complications in their incipiency; (5) uncorrected anemia; (6) prolongation of the operative time and excessive blood loss at operation; and (7) a general tendency to underestimate the possible risk, been appreciated by the attendants. Williams,[3] in a recent discussion of mortalities following excision of the uterus, states that he had the minimum preoperative requisites posted at the intern's desk on his gynecologic service in the Philadelphia General Hospital, and that he tried to have it followed. These requisites for all elective sections are:

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1. A complete history and general physical examination.
2. A leukocyte count of between 7,000 and 10,000.
3. A polymorphonuclear percentage of from 65 to 80.
4. A sedimentation time within normal limits.
5. A normal urinalysis, and an adequate kidney function.
6. A temperature of 98.6°F. for at least 48 hours before operation.
7. A systolic blood pressure between 110 and 150.
8. A negative Wassermann test.
9. Rest in bed in the hospital for a period of at least 48 hours, during which time the intake of fluids (water, milk) must be at least two and one-half quarts per diem, while the usual sugar intake of the individual must be at least quadrupled. Williams, in studying Polak's report of his mortalities, was impressed by the fact that the fatal issues were attributable in high proportion to omission or misinterpretation of history, to omission or inadequacy of physical examination or of laboratory procedure, or to disregard of some other important preoperative requisite.

Many weeks prior to elective surgery, the candidate should have all obvious foci of infection eradicated that can possibly be removed. Certain postoperative complications such as pyelitis and intravascular clotting are believed to be due in some instances to foci of infection in the teeth or tonsils, and might have been prevented by removing these foci before operation. Any patient over 40 years old should be given the most thorough examination of heart, lungs, liver, and kidneys; any abnormalities encountered should be given the benefit of consultation with an internist. An electrocardiogram should be routine in this age group. Elderly patients present an increased incidence of disease of these organs and since operative procedures add a considerable strain on the heart in particular, any degree of incompetency, however mild, may lead to serious trouble at the time of operation. Further, elderly patients should not be kept in recumbent position for long periods of time, but should be encouraged to moderate activity with intervals of rest. Latent, subsiding or quiescent respiratory disease may flare up following anesthesia of the inhalation type. A routine appraisal of the urinary system should include a catheterized specimen for urinalysis, P.S.P. and Fishberg concentration tests and blood chemistry to detect any degree of renal impairment. Disease of the liver should be looked for, utilizing the various hepatic function tests available (cephalin-cholesterol flocculation test and Quick hippuric test). When these tests indicate impaired liver function, intensive glucose therapy before and after surgery is indicated to improve liver function and spare this organ postoperatively. The use of decholin in addition has also been advocated. That glucose alone is not enough in these cases but protein is of equal or more value, to potentiate natural hepatic resistance is now being realized.

The value of adequate nutritional preoperative preparation, especially in patients who have sustained recent large weight losses, is recently stressed in an excellent article by Varco.[5] He states: "There is a broad zone existing between optimal nutrition and classical deficiency states in which abide many borderline cases of unsatisfactory dietary management and/or inadequate food intake." Adequate hydration, restoring of electrolyte balance, and adjusting the hemoglobin values by transfusion are not enough in patients who may also present a fatty liver and heptic dysfunction associated with, hypoproteinemia caused by malnutrition. These patients tolerate prolonged surgical manipulations, or even minor ones occasionally, poorly and may have a stormy convalescence or worse postoperatively. Varco has shown that such patients may be prepared satisfactorily for operation by feeding of high protein, carbohydrate, caloric, low fat diet.

We do not like to subject a patient to extensive surgery who has a red count of three and one-half million or less, or a hemoglobin of 70 per cent or less, unless this patient is transfused preoperatively. All subjects should have hematologic study, including the white and differential count, coagulation and bleeding time and a determination of the Rh factor. Any woman who is a Rh negative should receive of course only Rh negative blood if transfused. Typing and cross-matching is routine preoperatively and 500 c. c. of compatible citrated blood should accompany the patient to the operating room in all cases of major surgery, even if the blood count is normal. As stated above, the sedimenation rate and white count ideally should be within normal range before elective surgery; however a thorough bi-manual pelvic examination which does not excite a rise in temperature above normal is probably of more value in predicting whether the patient will have a stormy postoperative course or worse.

Particular attention should be given to obese subjects, in general poor candidates for operation. They are prone to develop pulmonary complications, acidosis, and wound infections. In addition, the technical difficulties at operation are much greater. If at all possible the obese woman should be placed on a weight regime.[6]

It has been noted that cases presenting large pedunculated submucous fibroids or polyps, partially blocking the cervical canal and obstructing drainage, are all actively or potentially infected. These growths should be removed vaginally, infection cleared up and hysterectomy delayed several months. Cases of this type are usually infected with a virulent type of streptococcus and hysterectomy is dangerous in their presence. Before this was recognized, fatalities due to this had been reported.

In all suspicious cervical lesions routine biopsy should be done before major surgical work. Early cervical carcinoma can be diagnosed only in this manner. Where biopsy reveals carcinoma, irradition, and not hysterectomy, would of course be the procedure of choice. All abnormal uterine bleeding near and particularly after menopause should be curetted for possible carcinoma before doing hysterectomy, because in carcinoma of the body of the uterus, preoperative irradiation followed by hysterectomy gives better results than either alone.

Before hysterectomy, all infected cervices should be cleared of as much infection as possible by douches, cauterization and other local treatment; otherwise an increased morbidity and mortality is to be expected. Estrogenic vaginal suppositories will facilitate plastic procedures on the vagina and combat local infection in cases presenting post menopausal atrophy of the mucosa. Preoperative administration of adrogens may make the surgical treatment of extensive endometriosis easier by causing regression of these abnormal growths. Cases presenting third degree lacerations of the perineum or recto-vaginal fistulae are best perpared by a non-residue diet and sulfasuccidine orally for a few days. Plastic procedures are best delayed until thorough involution has taken place following parturition, usually about three months. Exceptions to this rule are third degree lacerations and episiotomies that have broken down following initial repair at delivery.

All women to have vaginal surgery should have a series of acid douches peroperatively. All cases of vaginal or abdominal surgery are given a 1-10,000 potassium permanganate douche the night before and the morning of the operation and each douche is followed by the instillation of two ounces of a 4 per cent mercurochrome solution into the vagina. The bladder should be emptied by the insertion of a soft rubber catheter one hour before operation and it is taped to the thigh for continuous drainage, being left open during the operative procedure. One at times will see the abdomen opened for some pelvic surgical procedure in the presence of a half-filled urinary bladder, which may necessitate catheterization, interruption of the surgical procedure and loss of valuable time. The half-filled bladder may also be injured while making the usual suprapubic incision. All cases scheduled for vaginal surgery should also be prepared for a laparotomy because an emergency may arise necessitating entry into the abdomen for the control of hemorrhage or other complications.

A uterus may be punctured in performing a simple curretage and inaccessible bleeding may occur during a vaginal hysterectomy. Ureteral catheters should be inserted preoperatively in all cases presenting pelvic lesions which may displace the ureters and subject them to injury.

OPERATIVE TECHNIC

In choosing an anesthetic, each case must be considered individually. In general spinal should be reserved for the younger age group with normal cardiovascular systems. Most deaths from spinal anesthesia are circulatory in origin. It should not be selected in cases presenting extremes of blood pressure, that is, marked hypotension or hypertension, cardiac disease or advanced age or for patients with conditions associated with increased abdominal tension, such as pregnancy, ascities or large pelvic tumors. It is also contraindicated in cases of hemorrhage. Cyclopropane also should be used only in patients of the younger age group having normal cardiovascular systems. It is of especial value in cases of shock due to ruptured ectopic pregnancy. The injection of parasympathicomimetic drugs particularly pituitrin during cyclopropane anesthesia is to be avoided as fatalities have been reported.[7] Intravenous pentothal, in spite of its convenience in short procedures, is a dangerous drug presenting too narrow a margin between a therapeutic and lethal dose to recommend it for general use. Excessive uterine bleeding may be encountered in using it for anesthesia when doing a currettage for incomplete abortion. Ethylene and ether is still the anesthesia of choice in hypertension, cardiac disease and in the elderly. Were it always practical, the local infiltration of novocaine is near the ideal in so far as risk to the patient is concerned. Unfortunately it is not applicable in the majority of cases. In summary, all anesthetics are safest when administered by trained physician-anesthetists familiar with all indications, contraindications, and able to prevent or combat anesthetic complications, as they occur. In shock elevating the legs at right angles to the body is the simplest and most effective way of raising blood pressure quickly.[8]

While preparing the abdomen care should be exercised that an excess of ether, tincture of merthiolate, or whatever preparation is used, does not pool beneath the buttocks and back causing burns. This, combined with the forcible jerking of sheets or a Kelly pad beneath the patient, causes considerable skin damage.

An infusion of 5 per cent glucose is usually started after the patient is anesthetized. It is much better to have the needle in the vein when the blood pressure is adequate than to wait until the patient goes into shock, as collapsed veins are difficult to find. Citrated blood may be added as needed through the same apparatus.

Adequate abdominal incisions give better exposure and facilitate the operative procedure. Prevention of excessive blood loss, gentle handling of abdominal viscera, avoidance of undue trauma in the use of packs and retractors all help in the prevention of shock, postoperative distention and adhesions. Prolonged operations are associated with an increased incidence of complications and the procedure should be as short as circumstances allow. Routine appendectomy at the time of extensive pelvic surgery should not always be done as an increased mortality will result. Expression of air from the abdominal cavity as the peritoneum is being closed may help minimize postoperative discomfort and distention.

Postoperative care begins before the patient leaves the operating room. Approximately half of all anesthetic deaths occur in the immediate postanesthetic period, usually from asphyxia due to aspiration of vomitus, premature removal of intra-tracheal catheters, "swallowing of the tongue," or laryngeal spasm. The anesthetist should watch the patient closely until all reflexes have returned and the possibility of such complications has passed. The patient should also be closely watched after returning to her room by an experienced nurse until completely reacted. The blood pressure should be checked every half hour for three hours and shock therapy instituted if it falls to 90 or below systolic.

PREOPERATIVE CARE

We no longer, as in former years, purge the patient preoperatively and starve her postoperatively, resulting in the patient coming to and leaving the operation dehydrated and malnourished. Uncomplicated cases should get 2,000 to 2,500 c. c. of parenteral fluids during the first postoperative 24 hour period. Complicated cases (sepsis) require more (table 1). Tap water in small amounts, after nausea has subsided, may be given and deducted from the total fluid requirement. If abnormal losses are occurring they should be added as the amounts stated may be entirely inadequate.

TABLE 1

DAILY WATER REQUIREMENTS

Uncomplicated Case	
For vaporization	1,000 to 1,500 c.c.
For urine	1,000 c.c.
Total	2,000 to 2,500 c.c.
Complicated Case (Sepsis)	
For vaporization	2,000 c.c.
For urine	1,000 to 1,500 c.c.
Total	3,000 to 3,500 c.c.

In cases of dehydration as evidenced clinically by a hot, dry skin, dry tongue, fever and a scanty urinary output, an amount of fluids sufficient to maintain body fluids is not enough—an additional amount is necessary to provide for fluids previously lost (table 2). This large amount naturally would not be given during the first 24 hours and caution is necessary with old patients and those with cardiac deficiency. If the rate of administration be approximately 200 c. c. per hour and this is on a 24 hour basis by continuous intravenous drip the volume required is given.

TABLE 2

WATER REQUIRED FOR A DEHYDRATED PATIENT WEIGHING 60 KG.

Water for vaporization	2,000 c.c.
Water for urine	1,500 c.c.
Any abnormal loss	Variable
Water to restore depleted fuilds (6 per cent of 60 kg.)	3,600 c.c.
Total	7,100 c.c.

The average patient needs 5 to 6 grams of salt daily and 1,000 c. c. of normal saline solution contains 9 grams. Excessive administration of saline solution will produce salt retention and edema. Abnormal losses of fluids and electrolytes from vomiting, diarrhea, gastric and duodenal suction, intestinal obstruction, fistulae, shock, may occur. Fluids removed by gastric suction should be replaced volume for volume by saline solution (normal) or Ringer's solution. In cases of low plasma chlorides the giving of 0.5 grams of salt per kilogram of body weight for each 100 mg. that the plasma chloride has to be raised to reach the normal (560 mg. per cent) is a useful formula. Glucose, 5 per cent, is added to distilled water to make up the fluid requirements after providing for the electrolytes. One-sixth molar sodium lactate solution is indicated in severe acidosis. One-half is converted to liver-glycogen while the other half is oxidized to bicarbonate. This may be combined with Ringer's solution.

Hypoproteinemia may occur postoperatively being evidenced by peripheral and visceral edema, diminished intestinal motility and poor wound healing. The serum albumin range is 3.5 to 5.5 grams per cent while that of globulin is 1.5 to 3.5 grams per cent. The total serum protein averages 7.36 with a range of 6.2 to 8.1. Palpable edema is said to occur when the total serum proteins fall below 5.5 grams per cent or the albumin fraction below 3 grams per cent. Amino acids (amigen or parenamine) orally or parenterally may be used in an attempt to restore tissue proteins, plasma is indicated to restore serum proteins. However, as said previously, hypoproteinemia is better corrected preoperatively, if possible, by dietary means. Vitamins are necessary for patients receiving only intravenous alimentation and thiamin chloride 100 mg., nicotinic acid 50 mg., riboflavin 5 mg., and cevitamic acid 500 mg., may be added daily to infusions. Hypoproteinemia and a lack of vitamin C causes poor wound healing.

Normal cases postoperatively are usually able to take adequate fluids orally after the first 24 hours; however any deficit should be given parenterally. Few patients will take an adequate amount however, unless urged to do so at regular intervals by the attendants. The total fluid intake and urinary output should be accurately measured and charted on a separate sheet and added up every eight hours thereby giving at a glance this vital information. A soft diet is usually tolerated on the third postoperative day and a full on the fourth. Mineral oil is started at this time and an enema given on the fourth postoperative day in the usual case. A non-residue diet with the bowels kept constipated with opiates for at least a week is indicated in cases of third degree perineal repairs and recto-vaginal fistulae.

The postoperative care of the urinary system calls for regular catheterization usually at eight hour intervals until the patient is able to void spontaneously. Some prefer an indwelling catheter for the first two or three days. When catheters are used either indwelling or intermittently, small doses of sulfonamides are indicated as prophylaxis against infection. A check for residual is less than one ounce. The instillation of one ounce of ½ per cent aqueous mercurochrome after catheterization may aid the bladder to regain its tone. Daily urinalysis should be ordered on all cases receiving large doses of sulfonamides or transfusions postoperatively. Postoperative abdominal distention is minimized by the judicious use of morphine, rectal tube, heat tent and prostigmine. In cases of moderately troublesome distention these measures plus the turning of the patient over on her abdomen with the rectal tube inserted is often effective.

Cases presenting more marked distention and vomiting or those having had laparotomy or vaginal surgery with contamination or soiling of the peritoneal cavity, should be treated as for peritonitis with morphine, heat tent, rectal tube and Wagensteen suction with or without the Miller-Abbott tube. Sulfonamides and penicillin in full doses are also indicated both prophylactically in potential or actual cases of peritonitis, that is, in soiling of the peritoneal cavity at operation or in cases presenting signs of infection developing postoperatively. Oral feeding should be used cautiously in these cases. Crile,[9] of Cleveland, recently reported remarkable results in cases of localized or even generalized peritonitis, with the use of massive doses of penicillin; 100,000 units were given every two hours day and night for two days; then the dose was halved and given every two days, totaling four and one-half million units administered within a period of eight days. The combined use of penicillin and sulfonamides is often better than either alone as frequently we are dealing with a mixed infection, some organisms of which are controlled by one and some by the other drug.

Of the postoperative complications referable to the vascular system the first to appear are hemorrhage and shock. The latter is often noted in moderate degree soon after the patient is returned to her room and usually responds to intravenous fluids, heat and elevation of the foot of the bed. Plasma, blood and ephedrine may also be needed in severer cases as well as the use of nasal oxygen. Primary hemorrhage usually appears within the first six hours, possibly as late as 14 hours, and presents such findings as a falling blood-pressure and hematocrit and a rising pulse-rate, air hunger and shock. Early postoperative hemorrhage of severe degree requires location of the bleeding vessel and ligature, in addition to supportive measures.

Hemorrhage appearing later than the first 24 hours usually is secondary to a slough, particularly following vaginal hysterectomy about the tenth to fourteenth postoperative day. These cases following vaginal hysterectomy, which are by no means rare, usually respond to strict bed rest with the foot of the bed elevated. In more severe forms transfusions and even secondary suture of the cuff may be necessary.

The incidence of venous thrombosis and pulmonary embolism is claimed to be as high as three per cent of all laparotomies performed on women. Two types of intravenous clotting are well described by Alton Ochsner who has done much in recent years to give us a better understanding of this condition. A positive Homan's sign, and a low grade unexplained postoperative fever are very significant in making the diagnosis. Prophylactically, we may do much to prevent postoperative intravenous clotting, he says, by minimizing operative tissue trauma, dehydration, acidosis, intra-abdominal tension, tight bandaging, Fowler's position or Trendelenburg with the knees flexed, shallow breathing, inactivity while in bed, prolonged bed-rest following surgery, and eliminating foci of infection preoperatively. The common practice of placing a pillow under the popliteal spaces is to be avoided, and if a modified Fowler's position is needed, place and keep the thighs and legs in the same axis. The legs should be moved a "thousand times a day." Using the silk technic in suturing with early ambulation postoperatively is gaining much favor in recent years as a valuable prophylaxis against intravascular clotting postoperatively.

The application of dry heat by a light bulb to the perineum in vaginal surgery helps to relieve pain and prevent infection. Vulva pads are best omitted in such cases as they predispose to infection. The abdominal wound is not disturbed unless infection, if present as evidenced by tenderness, induration, pain and fever, is treated by early removal of skin sutures and by establishment of drainage if suppuration occurs. Finally, before the patient leaves the hospital, a check of all drains and packs used is made as it is at least embarrassing to have an overlooked vaginal pack discovered by the patient after she goes home. A printed list of instructions as to care after discharge saves a number of phone calls to the attending physician later and misunderstandings so common when only verbal orders are given.

SUMMARY

1. A plea is made for stricter application of the preoperative requisites of Polak before subjecting a woman to a major gynecologic surgical procedure. More postoperative mortalities are preventable if they are closely followed in elective surgical procedures. This was amply stressed by the investigation of the late Hilliard Miller in his review of deaths at Charity Hospital in New Orleans.

2. Thorough preoperative investigation of the heart, lungs, liver and kidneys should be routine.

3. The blood should be studied from the standpoint of total red and white count, hemoglobin, sedimentation rate, possible dyscrasias, and the Rh factor. Preoperative typing and cross matching is routine and compatible blood should accompany the patient to the operating room.

4. Special attention is required by the obese and the aged.

5. Local and remote foci of infection should be eradicated or minimized and dehydration, anemia, and avitaminosis corrected.

6. Each case should be individualized in choosing the proper anesthetic agent and all anesthetics are safest when administered by trained physician-anesthetists.

7. During surgery the administering of fluids, and at times blood, is vitally important. Avoidance of trauma, excessive blood loss and prolonged operating time minimizes postoperative morbidity and mortality.

8. Closely watching the patient until fully reacted from the anesthetic, control of pain, adequate fluid balance, alertness in detecting immediate or delayed postoperative complications in their incipiency are vital.

9. Adequate preoperative restoration of body proteins in a large group of patients with malnutrition by oral feeding of a high protein, carbohydrate, caloric, low fat diet is emphasized.

10. Finally, as stated by Hilliard Miller, no woman should die postoperatively from such avoidable errors as inadequate or delayed postoperative therapy, failure to detect postoperative complications in their incipiency; uncorrected anemia; undue prolongation of operating time and excessive blood loss at operation; and a general tendency to underestimate the possible risk involved.

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DISCUSSION

Dr. Alolph Jacobs (New Orleans): Dr. Johnson has covered a great deal of ground and it may seem very difficult to keep in mind all the indications, contraindications and precautions presented on the slides, but to those who do surgery these things come to mind as conditions arise.

I will discuss briefly one or two important points concerning contraindications in cases where there is a persistent low grade fever in degenerative uterine fibroids. One may wait forever for the temperature to become normal. Under these circumstances, low fever will persist, the sedimentation rate be rapid and a secondary anemia will be present. Operation therefore is indicated in order to eradicate the cause; proper supportive treatment being infusions and blood transfusions.

Secondary anemia in tubal abortion of an ectopic pregnancy will persist if unrecognized and not operated. Small spurts of blood occur and may persist for days. Under these conditions surgery is indicated in spite of the anemia, proper supportive treatment being given the patient.

Concerning urinary retention after surgery or delivery, it is a good practice to check for residual urine after the patient voids voluntarily for the first time, and continue to catheterize until there is no longer retention. Necessary precaution against bladder infection must be taken.

Dr. Ambrose Storck (New Orleans): One phase of the preparation of the patient going to operation which is very important in general surgical procedures and which often applies in the instance of gynecological patients is the estimation of transfusion requirements. In the past we have depended on hemoglobin determinations, red cell counts and hematocrit determinations, all of which are very undependable and inadequate. In an attempt to arrive at a more dependable means of determining transfusion requirements Dr. Champ Lyons has undertaken blood volume determinations and has shown that most of us are inclined to give too little blood. He uses a photoelectric colorimeter method which is comparatively simple and which any technician proficient in other laboratory procedure can learn to do with accuracy. Since we have been applying this guide in the preparation of patients for general surgery the results have been very gratifying. Many more patients have been getting the amount of blood they really need but would not have gotten if other guides had been depended upon. In gynecologic patients suffering from hemorrhage, infection or neoplasia the method is particularly applicable.

EXPERIENCE WITH SODIUM PENTOTHAL AT HOTEL DIEU

EDMOND SOUCHON, M. D.
NEW ORLEANS

Our introduction to intravenous anesthesia was very dramatic. It was thrust on us about ten years ago, at this hospital, in the form of a patient from DePaul Sanitarium, who had, in an attempt at suicide, used a Gillette blade with such enthusiasm on his own throat, that when he was placed on the operating table for inspection and his chin lifted up, the posterior pharyngeal wall was plainly visible. His incision extended from jugular to jugular (but did not sever or even nick these vessels or the carotoids), but proceeded very deeply the more mesially the incision extended. It was a jagged and irregular cut, but was remarkably free of bleeding. The only dressing that had been applied was a heavy bath towel which the patient himself held in position.

We were confronted with a serious problem as to the choice of anesthetic. Inhalation anesthesia was out of the question for mechanical reasons, spinal was out of the question because of the location of the injury, local was seriously considered but was discarded because of the patient's mental condition (he was constantly moving about in a highly excited state). The one choice we had was evipal or sodium amytal, intravenously administered, but no one had ever administered it at Hotel Dieu. We had no choice but to read the direction on the package rapidly and administer it ourselves, af-

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*Read before the Scientific meeting of the Orleans Parish Medical Society, June 10, 1946.

ter which procedure, we would race around to the field and begin the tedious attempt at rematching and suturing the numerous structures in the throat. The single dose of anesthetic would last between 30 and 45 minutes, after which time the patient would begin to move. Again the intravenous administration as repeated, again we would rush around and resume our repair. This procedure continued to take place for the three hours that it took to repair the wound, during which time it was necessary for us to resort to artificial respiration several times, and finally had to call Dr. Taquino to intubate the patient for fear of obstruction that was rapidly taking place due to edema.

This experience with intravenous anesthesia naturally etched itself deeply on my memory, and built itself up into a fear of anything that bore that label. Naturally, our conversion to sodium pentothal took longer and much more convincing that the average.

Our next experience with intravenous anesthesia was due entirely to one of the anesthetists employed at Hotel Dieu. I am referring to Miss Sim, and this was early in 1941. Of course, the first few cases were limited to practically nothing but minor cases, or cases that required a very short anesthesia. This group consisted of incision and drainage cases, dilation and curettage, setting of dislocations or fractures, and so forth. A few appendectomies were gradually introduced in our series. It was not until February, 1942, that we began seriously considering pentothal as the anesthetic of choice in practically any and all of our major and minor cases. This was when Mrs. Molly Seeberg became affiliated with this institution. She had been connected with the Hillman-Jefferson Hospital in Birmingham, Alabama, and this hospital was deeply investigating sodium pentothal over a period of years. Her experience and knowledge of this drug, plus the fact that she had given over 2000 anesthetics with it, was the final factor that led me deeply into its use.

HISTORICAL BACKGROUND

The historical background for intravenous anesthesia began with the use of chloral hydrate in 1872 by Ore of France. Following this, a group in Petrograd first used hedonal (methyl propyl carbinol urethane) in 1905. The volatile anesthetics were then used. Ether was given in saline solution intravenously by Burkhardt of Germany in 1909, and chloroform also had a brief trial. In this country Noel and Scuttar first used paraldehyde for this purpose. A brief trial was given ethyl alcohol by Marin of Mexico City in 1929. Avertin with amylene hydrate, first use in 1929 by Kirschner in Germany, had but little intravenous use. None of these received general recognition for intravenous anesthesia.

The first barbiturate to be administered intravenously was somnifen, a mixture of barbital and dial, and was introduced in 1924 in France by Fredet and Perlis. This preparation was subsequently changed to barbital and alurate. Pernoston was introduced by Bumm in Germany in 1927, and is still being occasionally used. It was probably the best intravenous anesthetic to be used prior to evipal. Following this sodium amytal was introduced in this country in 1929 by Zerfas. Pentobarbital sodium (nembutal), was tried in 1930, and found to produce a prolonged postoperative sleep. Evipal sodium was first used in Germany by Weese and Scharpff in 1932. Pentothal sodium was introduced by Lundy and Tovell in 1943. There have been a few barbiturates namely, soneryl, eunarcon, metyl luminal and thio-amytal which have been introduced in recent years, but which do not appear to have particular merit as intravenous anesthetics.

DESIRABILITY

Since the beginning of the anesthetic period, both the surgeon and anesthetist have been hoping to find the anesthetic agent which would produce rapid induction, quiet breathing, normal color, no salivary secretion and complete surgical relaxation; also one that would be rapidly eliminated and could be given with a maximum of safety—an agent that did not alter the function of the organs, and one from which recovery occurred without nausea or vomiting. Also one that the patient would accept without fear of apprehension were it necessary to administed a second and/or very proximal anesthetic. Our experience, although limited to approximately a series of but 1600 cases, has proved to our satisfaction that sodium pentothal possesses all these properties.

ADMINISTRATION

Sodium pentothal is a lemon yellow powder with a bitter taste associated with all the barbiturates. It has a sulphuric odor, and effervesces when mixed with water. A 2.5 per cent solution is made by mixing 1 gram of pentothal crystals in 40 c. c. of sterile distilled water. A blood pressure apparatus is placed on the arm (not to be used for venepuncture), and tucked close in by the side. The arm to be used for venepuncture is fastened securely on the arm board. A vein, preferably in the cubital fossa, is selected, a tourniquet applied, and a 20 gauge needle attached to a flexible sterile rubber tubing about six inches long, and again attached to a 10 of 20 c. c. syringe, is introduced in the vein, the tourniquet released, and the syringe containing the solution attached to the board by a clamp apparatus. Adhesive tape is used to keep the needle *in situ*, likewise the tubing. The skin may or may not be anesthetized with novocaine before introduction of the 20 gauge needle. Personally, we do not use it, as the one prick necessary to introduce the administering needle amounts to very little more than the prick necessary for the local anesthetic. From 5 to 10 c. c. of solution is slowly administered, taking about three to five mniutes for this introduction. The speed in which sodium pentothal is given is governed by the rate and depth of the respirations. After the above amount is introduced in the vein, it is best to pause for a few moments and carefully observe the individual reaction of the patient. We stress this point, because we have noticed that individuals of the same age, weight and approximately the same physical condition will react differently to the drug, some requiring a much larger dose and some a very small dose to produce the same degree of anesthesia. After a few minutes' pause, further administration of the anesthetic may continue. It is our experience that it takes but from 10 to 30 seconds for the patient to lose consciousness, and but three to five minutes more to be sufficiently relaxed for what we would term a deep surgical sleep where relaxation is complete.

It is our custom to administer oxygen throughout any surgical procedure. Our anesthetists are taught to introduce an airway as soon as abolition of the swallowing reflex or gagging is controlled, and this is done by further administration of pentothal if any resistance on the part of the patient is encountered at this point. Occasionally a nasal airway that extends into the trachea is used and oxygen administered in this manner. The eye signs are insignificant, except for the position of the eye. The pupil is rather small and sluggish in reaction to light. When the eye is not focused in the center it is, as with other anesthetics, indicative of light anesthesia; however, there may be enough relaxation for a major operation.

The depth and the rate of the respirations are the best signs to determine the depth of anesthesia. Very few patients need any further anesthesia after the peritoneum is closed. At the beginning of administration of pentothal, the following signs are noted.

1. Respiration at first may be stimulated to greater depth, followed by a very shallow respiration which persists throughout the administration. The rate, however, does not change. It is the depth of respiration.

2. The pulse rate may increase in rapidity at the beginning of induction, but will quickly regain the normal and remain so, granting that the surgical procedure or loss of blood is not a trying one.

3. There will be a drop in blood pressure of from 10 to 20 points in many patients, again with a return to the patient's normal after less than 10 minutes, granting that the surgical procedure or loss of blood is not a trying one.

SYSTEMIC EFFECTS

It was thought that pentothal sodium was contraindicated in cases of liver damage, but liver function tests by Quick hippuric acid test were done by Dr. B. M. Carraway of Birmingham, Alabama, in 100 cases, first doing the test before operation, and each day thereafter for 10 days. There was no drop in liver function and most of these tests were done on patients who were jaundiced or had liver damage. The same test was reported by Boyce and McFetridge in other anesthetics. On the first day ethylene showed at 21 per cent drop, ether a 25 per cent drop, and spinal a 49 per cent drop. None of these returned to normal until about the seventh to tenth postoperative day. Again, Dr. Carraway, in connection with Abbott Laboratories, performed extensive research on rats, rabbits, cats, dogs, and monkeys. One monkey was anesthetized under pentothal for 18 consecutive days, and his abdomen reopened each day, removing tissue from the liver for microscopic examination. He found no change in the liver at the end of 18 days, except some fatty infiltration, which is regarded as common for all types of anesthesia.

In another experiment that was carried out four dogs were used. All four were anesthetized with sodium pentothal, the abdomen opened and tissue removed for microscopic study. Two dogs were held as controls, the other two being anesthetized for a 20 minute period every 24 hours for 21 days. At the end of 21 days all four of the animals were sacrificed under sodium pentothal anesthesia. Except for fatty infiltration, there was no change in the liver cells of any of the dogs, and there was as much fatty infiltration in the controls as the other two.

The hemoglobin, erythrocytes, leukocytes, differential and urinalysis were run on a large series before, during and after operation, without any changes whatsoever. Blood chemistry was run before, during and after operation, also coagulation and bleeding time were made before, during and after (at 10 minute intervals during the operations), with no changes of consequence.

Electrocardiograms were recorded on 53 cases and reported by Dr. Edgar Hull. These tracings were taken some before, some during, some immediately after operation, and some one to 10 days after operation. There were no significant changes.

Kidney function tests were likewise run on a large series with negative reports as to damage.

THE PATIENT UNDER SODIUM PENTOTHAL DURING OPERATION

Much of the objection advanced by anesthetists and surgeons regarding the use of this drug during major abdominal operations was the lack of relaxation. In our series of cases we have found just the opposite, and relaxation is so complete that it compares very closely with that obtained with spinal. We believe that the reason for others, who employed this anesthetic, not obtaining the excellent relaxation that we have is due to the fear of both the surgeon and anesthetist to push pentothal to its maximum effect, consequently their patients were kept in a light plane of anesthesia, with resulting rigidity of the muscles and some straining. When pentothal was first introduced, a 10 per cent solution was used. This rapidly gave way to a 5 per sent solution. Personally, we have never used anything but a 2.5 per cent solution, allowing for a further margin of safety by the weaker dilution. We do admit, however, that our deep surgical anesthesia is pushed close to the minimal lethal dose of the drug, and for this reason never permit its use by any but an experienced anesthetist who has had special training with the administration of this drug.

Another feature that recommends itself to the operator is due to the shallow respirations that persist during its administration. This minimizes the intestines constantly forcing themselves into the field of operation (particularly good is this feature in cases where infected material is encountered) and consequently the infection is not spread as readily. We have stressed the point that oxygen is administered constantly throughout the operation to compensate for this shallow respiration.

Closure of the peritoneum is easily accomplished with a relaxation comparable to spinal, another feature which should recommend itself to the surgeon.

The use of the sulfonamides or penicillin in the abdominal cavity is not contraindicated with sodium pentothal.

Occasionally there will be hiccoughing, sneezing in some isolated cases. This was corrected by further administration of a small amount of pentothal, plus rebreathing the patient with carbon dioxide and oxygen. Usually, the preoperative administration of atropine will prevent this. However, if the stomach, upper intestines, or liver is handled too much or too roughly, it will produce these symptoms, which are much harder to overcome. The patient does not lose any fluid on the table because there is no sweating.

If for any reason it is desired that there be quick recovery from the anesthesia, 3 to 5 c. c. of coramine or metrazol is given intravenously. A few whiffs of straight carbon dioxide followed by straight oxygen may be given. Deep labored breathing will be produced by the carbon dioxide, and straight oxygen will give the patient an outstanding pink color. It is almost routine that the anesthetist give the patient 1 or 2 c. c. of metrazol or coramine, followed by oxygen after the peritoneum is closed. This will curtail the postoperative sleep. I have often questioned the advisability of this procedure if the condition of the patient is good, for what is the objection to a one or two hour sound sleep after the patient returns to his bed. On rare occasions, and we leave this decision entirely to the anesthetist, it will be necessary to supplement pentothal with nitrous oxide, an easy procedure since the patient already has a mask on. On still rarer occasions it has become necessary to switch to ethylene or cyclopropane, which is also easily done. Anesthetists who have given sufficient pentothal sodium anesthetics will develop confidence in it and learn that it is not necessary to administer it continuously during an operation, but only as the patient requires it. They also learn that the drug is somewhat cumulative in effect, and that the longer the operation, less and less of the drug will be required to keep the patient sufficiently relaxed. The amount to be given at the end of a two or three hours procedure is measured almost in the microscopic part of a gram.

POSTOPERTIVE RECOVERY

It is a well known fact that with pentothal the recovery to consciousness will almost invariably be in direct ratio with the length of the operation; that is, the longer the operation and the longer the drug is administered, the longer the patient will sleep after returning to his bed. In procedures up to one hour, an anesthetist who has had experience with the administration of this drug will have the patient beginning to spit out the airway and move about on the operating table. The very great majority of our cases, however, will begin to show signs of reacting in from one hour to two hours. There is usually a period of from 10 to 30 minutes when the patient will behave as most anesthetized patients do, and thrash about and attempt to sit up. Care is taken to instruct the nurses on the hall not to give the patient a postoperative hypodermic at this time. If left to go through this period of restlessness, the patient will relapse into a quiet and peaceful sleep for about two hours, after which time he will wake up quietly, like coming out of a natural sleep. After this period, it is safe for the nurse to administer a hypodermic for the relief of operative pain. If, however, a nurse becomes panicky at the first reaction of the patient, or does not know of the ill effects of a narcotic at this time and proceeds to administer morphia, pantopon, or dilaudid, he is liable to relapse into a deep surgical sleep for several hours, with possible impaired respiration and cyanosis. Unfortunately, the ideal postoperative procedure would be to have a nurse who is trained with patients reacting from this drug constantly on duty at the bedside until reaction is complete. It is also advisable to have at the patient's bedside a tank of oxygen-CO which should be administered through nasal catheter without excitement or alarm—in contrast to the present procedure which calls for pandemonium and great fright to the relatives of the patient sitting around the bed should oxygen be required at a moment's notice.

Again, the nurses on the hall become very alarmed at the shallowness of the respiration that continues for a while, but they should be taught that if the respiratory rate and the patient's color are normal, there is no need for alarm.

One of the features that recommends itself to those who use it and those who receive it is the absence of postoperative vomiting. It is our usual instruction that the patient may nourish as often as desired, as soon as reaction is complete, and if the operation itself does not contraindicate. At first we permit small amounts of surgical liquids, but that same afternoon nourishment may be given freely as the patient desires. We find that usually a single 1500 c. c. infusion of 5 per cent glucose is greatly beneficial, because the one peculiar reaction that a patient usually complains of is intense hunger, which we believe is due to a using up of the glycogen supply in the liver and this glucose, plus the usual surgical liquids, quickly replaces this deficiency. This is purely a conjecture on my part, however.

I have noticed a markedly reduced amount of "gas pains" and postoperative distention since using this drug.

Whatever postoperative nausea or vomiting is encountered I believe sincerely is due to the patient's idiosyncrasy for the opiates, or from an overzealous nurse who will overnourish the patient for the operator's benefit.

Occasionally some patients will have double vision or an inability to focus for 48 hours. Reviewing the literature on the subject, we are unable to find any explanation for this phenomenon. In no case, however, has it ever persisted over two days.

CONTRAINDICATIONS

The literature on this subject is controversial, but is gradually leaning to the very enthusiastic view that we ourselves carry. Extreme youth and senility formerly headed the list. In our limited series, the youngest patient in whom pentothal was used was four years old, and the oldest 83. Both had gangrenous appendices, and made uneventful recoveries. I can see, however, in senility that a prolonged sleep if an overdose of the drug were given would certainly make for a good start in a hypostatic pneumonia. In infants, naturally the dose is very much smaller, the big objection being the smallness of the veins and the difficulty of introducing the needle.

Operations on the face and in the throat, where much mucous is liable to form and collect if the vomiting reflex is absent for a prolonged period, would naturally predispose for aspiration with the resulting aspiration pneumonia and lung abscess.

We have used this anesthetic with good results on three patients with bad appendices who were also in the midst of a severe bronchial asthmatic attack. We have also used it in at least 50 major laparotomies in patients who had severe heart murmurs. Of course, the hearts were compensated at the time of operation, or this anesthetic would not have been attempted.

Cases of pneumonia are definitely a contraindication, but does not this condition cause great concern in the selection of any of the other anesthetics?

Hyperthyroidism is another contraindication, because of the rapidity with which detoxification of the anesthetic is accomplished, making the administration a difficult problem for the anesthetist.

Myxedema, where the reverse process exists, and where detoxification is retarded and there is likely to be a cumulative effect due to a slow detoxification, also contraindicates the use of this drug.

Operations on the intestines do not lend themselves readily to this anesthetic, due to the reflex action causing hiccoughing or sneezing. I do believe, however, this will be overcome at a later date.

In operations of great length there is a wide divergence of opinion in the literature, both sides being equally as emphatic. It is my personal belief that with an experienced anesthetist the length of time should not be considered.

Alcoholism may be regarded as a contraindication due to disturbed basal metabolism, also to liver damage, and also to the possibility of hypostasis. Also, there is a tolerance to the drug, requiring much greater amounts to be administered, with the possibility of a very sudden cumulative effect, unto death.

In our series, we have not chosen our cases, and all those except where a definite contraindication was noted, we have administered it routinely. Our cases have included innumerable appendectomies, a large number of hysterectomies, an equally large number of pelvic cases (tubes, ovaries, cyst), hernias, several gallbladder, tubal pregnancies, plastic repairs of the perineum and cervix, vaginal hysterectomies, breast cases, hemorrhoidectomies, and one classical cesarean section. Incidentally, this one and only cesarean section with pentothal was ideal from the maternal point, but it took us almost an hour to revive the infant.

Although the literature tells us to the contrary, I am very hesitant to use pentothal on patients who are debilitated, or who have been exsanguinated, or with a marked hypotension, due to the fact that pentothal almost invariably produces a drop in pressure at the onset of administration. In reviewing the few cases that have given us considerable concern on the table, it was invariably this type, and insufficient rehabilitating preoperative measures had been used. We have had but three cases in this series that required resuscitating measures on the table, and I believe that in retrospect, I was more frightened than familiar with the true condition of the patient. Artificial respiration, plus coramine or ephedrin, plus oxygen-carbon dioxide forced into the patient's lungs brought about a satisfactory response and it was possible to continue the operative procedure. Two of these cases occurred before the abdomen was opened, the third after two hours of hard surgery in the pelvis, while Dr. Mouledous had one case to cause concern after being returned to his room (we doubt that the true condition of the patient was recognized).

SUMMARY

1. Sodium pentothal is a non-irritating anesthetic, giving a smooth and rapid induction.

2. The pulse, respiration, and blood pressure are more nearly normal than with other anesthetics.

3. The color is pink and the skin warm and dry, due to no loss of body fluids.

4. Excellent surgical relaxation is obtained when properly given.

5. No nausea or vomiting are present except when the patient is susceptible to the narcotic.

6. Pentothal is very pleasant from the standpoint of the patient, and there is no dread of repeated anesthetic.

7. It is a safe anesthetic when administered by an anesthetist who is familiar with its action.

OTHER USES OF SODIUM PENTOTHAL

1. To supplement local, spinal, or caudal anesthesia.

2. To combat toxic effect of a local anesthetic. Should a reaction occur, inject 2 to 4 c.c. of a 4 per cent solution. This will counteract particularly the toxic effects of cocaine or its derivatives.

3. To relieve convulsive states that occur with drug poisoning; tetanus, eclampsia. ether anesthesia, and so on.

4. Occasionally used as a "truth serum."

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DISCUSSION

Dr. Louis Levy (New Orleans): Dr. Souchon is to be congratulated on bringing before the Orleans Parish Medical Society the timely paper on sodium pentothal as an anesthetic given intravenously.

I have been fortunate enough to peruse this paper with the idea, if possible, to add to his fund of knowledge of sodium pentothal and the technic of administration. I can but commend the author and advise strict adherence to his detailed instructions.

It is interesting to note the advance in anesthesia. In about 1910, I reported 1512 cases of ether anesthesia, without an anesthetic death. Now that I have nearly discarded ether as an anesthetic, I can report no less than 200 cases of sodium pentothal anesthesia without a death from the anesthetic. I was first impressed with the articles appearing in the journals and then by seeing Dr. Souchon use it on several cases before I decided to use it.

I am, in experimental work, "not the first to go or the last to follow", and I have used this anesthetic after fully deciding it was a safe one.

The advantages and cautions in its use to get the best results are worth emphasizing:

1. Have your patient calm; give the usual sedative preoperatively.
2. Have an anesthetist skilled in the use of intravenous anesthesia.
3. Use oxygen as an adjunct, as shallow respirations are one of the effects of this anasthesia.
4. Operations lasting two and one half hours are best continued with gas anesthesia.
5. The anesthetist should be relied on to announce the maximum dose, deciding by the time of operation the condition of the patient and the severity of the operation.

Some superior points in and after administration are:

1. Ease of administration.
2. Perfect relaxation: I have described "the abdomen as a wet paper bag" during this anesthetic.
3. The patient sleeps as though in a state of suspended animation; and, unless long operations, loss of blood, or rough handling interfere, awakens as from natural sleep.
4. Allowing your patient to sleep and not be awakened by the use of antagonistic drugs; such as, metrazol, is of great benefit to the patient.
5. Catch the awakening pains by the use of a narcotic so that the frightening ordeal of surgery will not be evident; the narcotic is begun as soon as restlessness appears.
6. An airway left in situ eliminates the dangers of tongue swallowing and can be removed as soon as signs of awakening appear.

I have noted in the literature that curare is being used with sodium pentothal to increase relaxation; I have yet to need an additional drug to induce increased relaxation in the administration of sodium pentothal.

I feel that the Orleans Parish Medical Society and Hotel Dieu are indebted to Dr. Souchon for presenting his valuable paper on sodium pentothal anesthesia.

Dr. Dean H. Echols (New Orleans): Fortunately some of our surgeons take the trouble to interest themselves in anesthesia. I have just one small thing to add, namely, that pentothal has proved to be just as important in neurosurgery as in Dr. Souchon's work. I estimate that 60 per cent of the neurosurgical procedures which we have carried out in the past two years have been done under pentothal anesthesia.

INTRAVENOUS PYELOGRAPHY AS AN AID IN DIAGNOSIS*

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Intravenous pyelograhy or urography has been in general use long enough now to enable one to gauge its indication and value. In infants and children it avoids the necessity of a general anesthetic and the accompanying radiographic difficulties. In these small patients when the intravenous method is impossible the dye may be injected intramuscularly or subcutaneously with excellent results. In the aged or in hypersensitive patients or in some anomalies or obstructions of the urinary tract distal to the bladder the intravenous or excretory method may solve your problem.

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*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May6-8, 1946.

While the character of the shadows of biliary calculi, phleboliths, calcified mesenteric glands or other calcifications outside the urinary tract are generally distinctive on a plain K. U. B., they can at times be annoyingly similar to urinary calculi. The intravenous pyelogram will aid considerably and often conclusively in their differentiation.

The intravenous pyelogram will give you the size, location and contour of the pelves, calices and ureters. Films made in various positions will demonstrate ptosis or displacements of the urinary system, hydronephrosis, kinking of the ureters or at times aberrant vessels. Anomalies of the urinary system are demonstrated. In deciding whether an abdominal mass arises from the urinary tract the intravenous pyelogram may decide the issue by demonstrating a normal tract in normal position, thus excluding the mass as of renal origin; or it may show the mass to be a normal kidney abnormally placed; or, too, it may show the pathology to be definitely arising from a portion of the urinary tract. However, when a suspected renal tumor is demonstrated by intravenous methods a retrograde examination should be done for confirmation.

Right here it may be emphasized that the intravenous pyelogram can never replace cystoscopy and retrograde pyelography. Anyone even moderately well informed will realize there are too many procedures other than radiography which can be done during retrograde pyelography to allow us to accept intravenous pyelography as a complete substitute.

The technic for intravenous or excretory urography is relatively simple. It can with proper supervision be done in the office although all ours are done in the hospital probably as a matter of convenience. The patient is prepared as for an ordinary K. U. B. examination, with this addition; no fluids are allowed after the evening meal preceding the day of examination. This is to obtain a better concentration of the dye in the urine. The morning of the examination a scout film is made. If it shows an over amount of gas in the intestinal tract is it better to delay the examination until the condition is corrected. This may be accomplished by the use of castor oil and enemas with postponement of 24 hours. At times enemas or pitressen (if not contraindicated) may clear the condition in about one hour. Twenty or thirty c. c. of skiodan or a similar preparation is administered slowly. Routinely films are made 5, 15, and 30 minutes after completion of the injection. One upright projection should be made and oblique one when indicated. When there is hydronephrosis of marked extent, film studies should be delayed a matter of hours.

Contraindications are listed as liver disorders, nephritis, uremia and most important one the iodine sensitive patients. We have encountered no difficulties ourselves although fatalities have been reported.

CONCLUSION

We wish to state that in our experience the intravenous pyelogram has proved extremely valuable as will be shown in the demonstration of some lesions by lantern slides.

BIOLOGICALLY FALSE-POSITIVE WASSERMANN TESTS IN NON-SYPHILITIC PATIENTS*

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Thirty years ago the interpretation of results of serologic studies was simple—a positive or negative report of a single test was generally accepted at its face value in the diagnosis of syphilis. Today the problem is entirely different. Using a battery of two to eight technics, the result of each technic is reported in one of six accepted terms, and the report cannot be applied without clinical interpretation.[10] The necessity exists of all physicians understanding the limitations of the serologic tests for syphilis, as well as having ready knowledge of the variety of conditions that have been found to produce biologic false-positive serologic tests.

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The social stigma that is attached to syphilis causes a psychologic trauma which obviously demands considerable effort to avoid an erroneous diagnosis. During the past war this problem also had great military importance.

Even with the present improved standardization, there is still considerable instability of the sensitivity of a given procedure, so that conflicting reports between different laboratories, or between successive tests in the same laboratories, are frequently encountered with syphilitic sera of border line reactivity. In view of the possibility of excessive sensitivity, as well as the possibility of clerical errors, a single positive report should not be considered valid evidence of syphilis or of a biologic false-positive reaction.[4]

The criteria that is generally accepted for the term biologic false-positive Wassermann is that the serum is positive to one or more types of tests, but which becomes negative after a few weeks or months without antisyphilitic treatment. The reliability of these cases depends on the reliability of the laboratory which performs the tests and upon the demonstration of more than one positive blood specimen before the tests become negative, in order to eliminate suspicion of a clinical or technical error.

Serodiagnosis of syphilis was inaugurated by Wassermann, Neisser, and Brunk (1906) with their complement fixation tests. Soon after, Michaelis, using a similar antigen, the saline extract of syphilitic liver, outlined a method for producing visible precipitates with syphilitic serum, thus describing the first precipitation reaction. Following this it was discovered that extracts of tissues not containing *Treponema pallidum* are satisfactory antigens for complement fixation and precipitation tests, and that alcoholic extracts of organs from normal animals make more satisfactory antigen than aqueous extracts of syphilitic organs as used previously. These alcoholic extracts of normal tissue contain the lipoid substances. Even though these lipoid extracts are not true antigens in a biologic sense, they react in an extraordinary specific manner with syphilitic serum. There have been numerous hypotheses regarding the nature of syphilitic reagins and the mechanism of non-specific lipoid antigens in producing positive serologic reactions for syphilis. At present, none of the proposed theories fits all the facts.

Numerous modified methods of complement fixation testing have been described and accepted, but it was not until 1922 that the great possibilities of flocculation methods were demonstrated. The main advantage of the first flocculation tests was their apparent simplicity. Various flocculation tests had by 1928 reached such degrees of sensitivity and specificity that, following comparison with various accepted complement fixation tests at the Second Laboratory Conference of the League of Nations held in Copenhagen, the better flocculation tests were considered equal to the better Wassermann tests. Results similar to the above were obtained at the Montevideo Conference (1930), where the best flocculation tests were demonstrated to be superior in sensitiveness and equal in specificity to the Bordet-Wassermann reaction and its modifications. The results of the American Serological Conference (1935), in which serodiagnostic tests for syphilis used in the United States were evaluated, indicating that efficient flocculation and efficient complement fixation tests for blood or spinal fluid specimens are of about equal value.

Additional conferences held in the United States have shown that at least five complement fixation and flocculation tests give satisfactory results when performed by persons other than the originators.[16] These are the Eagle, Hinton, Kahn, Kline, and Kolmer tests, the technics of which will not be discussed here. However, the Kahn verification test and the Gaehtgens *Treponema pallidum* (Reiter strain) complement fixation test, because of their possible future value in distinguishing between true and false-positive serologic reactions, will be discussed.

Kahn in attempting to identify false-positive serologic reactions in human beings, conducted studies concerning the differentiation between the reaction in apparently normal animals and in known cases of syphilis. These studies led to the observation that the temperatures at which serologic tests are carried out provide a means of differentiation. Various experiments were carried out including tests at different temperatures on normal animal serum, non-syphilitic human serum, and syphilitic human serum. The precipitation technic is carried out at 37° C. and 1° C. Serums that give positive reactions with a diagnostic test are examined with this procedure, and the precipitation results noted. The result is reported "positive" (syphilitic type of reaction) when precipitation occurs at 37° C. with practically negative results at 1° C. The result is reported "negative" (general biologic type of reaction) when precipitation occurs at 1° C. with practically negative results at 37° C.

Kahn states that the verification test is intended to be used as a supplementary procedure to diagnostic serologic tests when such tests give doubtful, weakly, temporary, or fluctuating positive reactions in cases of asymptomatic syphilis. If the results of the verification test correspond to those of the diagnostic test, they may be interpreted as corroborating the results of the latter test. If the results of the verification tests are negative, the probability is that the reactions given by the diagnostic test are non-specific. The major function of the verification test is to help reduce, and not increase, the number of cases of "serologic syphilis," therefore the test should be utilized as a supplementary procedure primarily with the diagnostic test and not with supersensitive tests.[8] The extent to which the verification test can be employed to verify the specificity of supersensitive tests is yet to be determined. Some observers, namely Chargin, Rein, and Schaffer, find it to be quite trustworthy, while others, namely O'Leary and Schoch report repeated failures.[19]

In 1929 Gaehtgens reported that syphilitic serums gave positive complement fixation with phenolized cultures of *Treponema pallidum* (Reiter strain) and recommended the use of this phenomenon for the serodiagnosis of syphilis. There have since been numerous reports, most of them from German laboratories, which have confirmed the practical utility of the test as recommended by Gaehtgens. Many of the reports indicated that the reaction is more sensitive than the Wassermann or flocculation tests with tissue lipoid, and its greater specificity has been repeatedly stressed.

In 1940 Erickson and Eagle carried out a study of the practical utility of the spirochetal complement fixation on 1,032 specimens, in combination with the icebox Wassermann test and the Eagle microflocculation procedure. In carrying out these tests spirochetal suspension was simply substituted for ordinary Wassermann antigen. Of the 1,032 serums examined 490 were from patients under treatment for syphillis and were used as a check on sensitivity; 542 serums from patients of a general dispensary and hospital population who had previously been found to have negative macro-flocculation tests, were used as a check for specificity.

In the syphilitic group—the proportion of positive results with the Wassermann, flocculation and spirochete technics were 57, 67, and 74.5 per cent respectively; while the total serums detected (positive and doubtful) were 68.5, 76.5 and 85 per cent respectively. Moreover, of the 436 serums which were detected by some one of the three tests used in combination, only 20 were missed by the spirochetal complement fixation test, as compared with 63 for the flocculation test and 83 for the Wassermann. The superiority of the test using cultured spirochetes as antigen is manifest. The high proportion of serums detected is particularly noteworthy since these serums were collected from patients who, on the average, had been given significant amounts of anti-syphilitic treatment.

As to the control group of 542 flocculation negative patients, 522 were negative to both the Wassermann and spirochetal technics. Of the remaining 20 there was one whose reaction was positive to the Wassermann test and negative to the spirochetal complement fixation test during an attack of jaundice; when the jaundice disappeared, the Wassermann reaction became negative. A second case, positive to Wassermann and spirochetal fixation tests, was known to have congenital syphilis. There remained for analysis 18 cases in which spirochetal complement fixation reaction was positive or doubtful, in the face of a negative Wassermann and negative flocculation tests. On clinical recheck from standpoint of syphilitic infection, repeat serologic tests, or both, were feasible in 17 of the 18 patients. The data on these patients show that a definite diagnosis could be made on the basis of history, clinical findings or repeat serologic tests in no less than 14 of the 17 cases, and in at least one of remaining three there was strong presumptive evidence of syphilitic infection.

From the above it is evident that the Gaehtgen's test gives promise of being the most important serologic step since the Wassermann discovery, even though at present it has a very narrow useful range (1:6 to 1:8), being anticomplementary in dilutions 1:2 to 1:3 and extremely weakened in dilutions of 1:10, and that it is a proprietary preparation.[6]

With the assumption that the routine Wassermann like reactions are understood, the possible mechanisms of false-positive tests will be considered. It is conceivable that there may be present in the serum: (1) an antibody identical with the Wassermann antibody produced in syphilis; (2) an antibody differing in some way from time to time with true Wassermann antibody but which cross reacts with Wassermann antigen or some component thereof, and (3) physio- chemical changes, not involving antibodies, which produce flocculation or complement fixation reactions.

If the antibody is formed in response to Wassermann antigen released from the host's tissues in a suitable form, rather than to spirochetal antigen, it would not be surprising if other disease agents occasionally produce the same reaction. In such cases the antibodies would be identical and no possible improvement in the serologic tests for Wassermann antibody could eliminate false-positive biologic reactions. There is no way of knowing at present what proportion, if any, of non-syphilitic positive tests belong to this category.[4] It has been shown by Lund that in supposedly normal serums that there is a very low titer of Wassermann antigen. This was demonstrated only after increasing the ratio of serum to antigen approximately ten times those used in diagnostic tests. Malloy and Kahn have been able also to show that normal sera contains some Wassermann reagin. It is suggested that occasionally a normal human serum may contain enough of this reagin to give a positive test for syphilis. Eagle in 1941 reported a serologic study on 40,545 white college students of both sexes. Sixty-two of these were reported to have positive or doubtful positive serologic tests (40 positive—22 doubtful). Of the doubtful cases it was satisfactorily estimated that an incidence of only 1:4000 existed. Statistically this result can be regarded as negative, but to each individual case the interpretation is of the utmost importance.[1a]

The main hope of the problem of biologic false-positive biologic Wassermann seems to fall in Group 2 above. Several examples of a similar phenomenon are in evidence today. The best example in use today is that of the heterophile sheep cell agglutination tests used in infectious mononucleosis. No definite evidence is yet available concerning the possibility of this described cross reacting, although different type antibody.

Group 3 above cannot be adequately disproved. It has been stated that alteration in blood cholesterol and blood lipid content may play a role in false biologic positive Wassermann reactions especially in pregnancy and jaundice, where these substances are elevated. Results of studies done by Cardon and Atlas (1942) seemed to indicate that a condition of hyperproteinemia is not associated with false-positive reactions. The authors state, however, that their series of cases is too small to make adequate conclusions and propose that in cases of false-positive tests that the total protein and serum globulin concentrations be determined when there is reason to suspect such a reaction.[9] No other adequate data are available concerning this group.

Various other possible explanations have been proposed in regards to conditions causing false-positive biologic reactions, and therefore will be considered under each disease or cause discussed.

INCIDENCE IN VARIOUS DISEASES

Malaria: Dawber has pointed out that malaria may be one of, if not the chief cause of false-positive Wassermann reactions. This condition is quite common and evidence has been presented that it is often present in a subclinical state; particularly chronic and latent malaria may produce positive tests which may easily be mistaken for latent syphilis and that the distinction between the two will not always be easy. At the Maine hospital, in Norfolk, Virginia from 1936 to 1940 there were a total number of 64 cases of malaria, with a total of 19 cases with positive serologic tests. Seven of these were definitely diagnosed as syphilis, four probably due to syphilis, and eight with false-positive serologic reactions. From a study of these cases it is concluded that if one allows a month to elaspe following a malarial infection, the serologic reactions should certainly have become negative, assuming that adequate malarial therapy had been given.[5]

In the 1935 Evaluation Survey 36 nonsyphilitic patients in various stages of malaria gave 11 to 19 per cent positive results with various tests. A more extensive study, by the same committee, of 266 patients found that only 4 per cent gave positive and doubtful Hinton, Kline diagnostic and Kolmer tests.[4]

Eagle found some degree of transient reactions to the Wassermann or Eagle flocculation test in nine of eleven cases inoculated with Plasmodium vivax.

Potter, Bronstein and Gruber point out that malaria will be one of the post-war problems, but also emphasizes that the problem in relation to false biologic positive Wassermann reactions is not considered by most physicians. In a well defined controlled study they took blood and spinal fluid form 100 consecutive patients with malaria within the first 48 hours of entrance to the hospital. These 100 patients gave no history of syphilis and none had had syphilis registers opened on them. Those who had positive or doubtful blood tests had repeat tests done about once a week until negative results were obtained. Twelve per cent had positive tests, and 10 per cent doubtful tests. The spinal fluid Wassermann reactions were negative in all cases, so it is suggested that the factors which produced the biologic false results in the blood are not present in the spinal fluid, although the spinal fluid is of no value in judging whether or not the blood serologic reactions indicate the presence of syphilitic infections. All serologic reactions of these 22 patients were negative within one month, although no definite conclusion is reached on the length of time required for the reaction to become negative. It is pointed out, however, that 10 of the 22 soldiers had a change in the Wassermann reaction without a change in the Kahn reaction, which fits in with statements by Eagle and Mohr that serums that are positive with sensitive tests and negative with more sensitive ones are probably not syphilitic.[14]

It can be concluded that although there is definite evidence of the occurrence of false positive test with malaria, that there is, however, need for further study concerning the mechanism for reaction, and average time length of persistence of the reaction.

Infectious Mononucleosis: The first case reported of false serologic tests in this condition, was by Lőhe and Rosenfeld[4] in 1928. Hatz in 1938 published one clear-cut case with strongly positive transient Wassermann reactions and simultaneously negative Kline tests.

Bernstein in 1938 reported six cases in

which the Wassermann and the Eagle flocculation test, or both, became temporarily positive during the course of infectious mononucleosis.

The incidence of false-positive serologic reactions in infectious mononucleosis is difficult to determine with any degree of accuracy. According to the combined series of Goding, Saphir, Sudusk, Bernstein, and Kaufman, there were 29 patients with false-positive reactions out of 183 patients with infectious mononucleosis who had tests for syphilis performed.

It has been pointed out by instances reported in the literature that individual cases of infectious mononucleosis with false-positive serologic tests fail to reveal anything characteristic about the disease in clinical course, the blood count or smears, or the sheep cell agglutination tests. There does not seem to be any quantitative relationship betwen the heterophile antibodies, which give rise to the Paul-Bunnel reaction, and the substances responsible for the positive complement-fixation tests or the various flocculation tests (Kline, Kahn, Eagle). False-positive reactions do not seem to be more frequently encountered with one serologic test than with another, except in so far as certain tests, such as the Wassermann are more commonly used.[9]

Yaws: This disease is known by all to be caused by a spirochete which is morphologically almost indistinguishable from *Treponema pallidium* and shares many of the clinical characteristics of the disease. Wassermann serologic reactions are known to occur regularly with this disease, and may or may not be considered as a false-positive biologic reaction. Moore states that the incidence is 100 per cent with this disease.[2]

Tuberculosis: As yet there has been no definite conclusion reached concerning whether tuberculosis actually gives rise to false-positive biologic reactions. However, if it did in even a small percentage, this would be significant in so widespread and chronic a disease. Davis agrees with Sweany who has reported that in 1000 autopsies of patients dying of tuberculosis, pathologic signs of syphilis were found in one-third of the 4.1 per cent patients with a positive Wassermann reaction. Inasmuch as it is known that negative autopsies are not unusual in old syphilitics, he concluded that there is "no reason to warrant the suspicion that tuberculosis causes any more than an insignificant number of false reactions,"[4]

Trypanosomiasis and Kala-azar: Early experimental trypanosomal infection of rats and rabbits was reported to cause positive Wassermann and flocculation reactions, but it was found later that even normal rabbits gave at times a positive reaction. No definite evidence has been presented within the last ten years that clinical trypanosomiasis produces false serologic reactions.

Various conflicting reports are available concerning false serologic reports, but it is stated by Davis that the laboratory work in tropical countries is, in general, not adequately standardized and that the chronic nature of the disease prevents the application of the criterion that the reactions be transient.

Leprosy: It has long been known that leprosy can produce positive biologic reactions, which are reported as being quite frequent. In the 1935 Evaluation Survey 50 leprous serums were examined and it was found that the incidence among five accepted tests varied from 40 per cent with the Hinton to 72 per cent with the Eagle flocculation test.[4] Moore states that the incidence is between 40 and 80 per cent.[2]

Due to the fact that this disease is rare in the United States there is little need for serious concern by the practicing physician.

Vaccination and Immunization: According to various reports, biologic false-positive reactions occur with some frequency following smallpox vaccination. A recent control study, by Rein and Elsberg (Army Medical School), seems to confirm previous reports and will be considered in some detail since this is a most important problem.[15]

The series were adequately controlled, using only non-positive reactors at time of smallpox vaccination (tetanus and typhoid immunization also given). All specimens were subjected to Kline diagnostic, Kline exclusion, Boerner-Jones-Lukens, Mazzini and Kahn standard flocculation tests, and Kolmer complement fixation test. An attempt was made to examine each subject serologically at weekly intervals.

"The types of skin reactions due to the vaccine virus were classified into three groups:

(1) Vaccines reaction (primary take);

(2) Vaccinoid reaction (secondary or accelerated reaction);

(3) Immune reaction (immediate reaction).

"One hundred twenty-nine individuals developed a vaccinoid or vaccinia reaction following smallpox vaccination, of whom 58 (44.9 per cent) showed doubtful or positive tests. In this group of serologic reactors 50 (86.2 per cent) developed their first positive serologic reaction between the eighth to fourteenth day following the vaccination. Three individuals developed the first positive serologic reaction on or before the seventh day, and 5 individuals developed the first positive reaction between the fifteenth and twenty-first day following the vaccination.

"Eighty individuals developed vaccinia reactions of whom 41 (51.2 per cent) showed false-positive reactions, whereas 49 individuals developed vaccinoid reactions, of whom 17 (34.7 per cent) showed false-positive serologic reactions. This would seem to indicate that false-positive serologic tests occur more frequently in patients in the vaccinia group than in the vaccinoid group."

Thirty-six individuals, who developed immune reactions, had an incidence of 13 per cent serologic reactions. Two appeared in the first week, none in the second week, two in the third and one in the fourth week following vaccination. These serum reactions usually disappeared in one week. The latter immune reactor series is too small to draw any conclusions.[15]

Various percentage figures have been given concerning the frequency with which false-positive reactions occur with smallpox vaccinations, and range from 11 to 51 per cent.

It has been shown by Arthur and Hale that on 95 soldiers who had received smallpox vaccination, typhoid injection, one injection of yellow fever vaccine and three tetanus injections within a three month period, that 14.8 per cent of these men had a temporary change in the normal negative Kahn reaction. This change persisted for one to two months and then faded out. He points out that before a diagnosis of syphilis should be made after routine immunizations that at least one to two months period should elaspe.[1]

Heimoff in 1944 reported a series of eight people with biologic false-positives, in whom only a recent stimulating dose to tetanus toxoid was a common factor. These eight persons all had negative histories of syphilis, all maintained a positive serologic test for six to twelve weeks, and all reversed to negative without treatment.[7]

From the above considerations, although not definitely conclusive, it is evident that smallpox and tetanus toxoid vaccinations, should always be kept in mind when serologic tests for syphilis, without history or physical findings of syphilis are positive.

Rat Bite Fever: There is a slight morphological resemblance between *Spirillum minus* and *Treponema pallidum*, although in only a few cases was it ever proven to be the etiological agent of this disease.[4a] Seventeen cases have been reported in the literature since 1930 but only 10 had positive Wassermann or flocculation tests.

In 8 cases tested serologically in which *Streptobacillus moniliformus* had been recovered from the blood stream by culture, three had positive tests for syphilis. One probably had congenital syphilis, the other two were laboratory injections (no follow up on these cases).[4] From the evidence presented in other reports, no definite conclusion can be reached as to whether false-positive biologic Wassermann reactions occur with injection with this organism. In a milk borne epidemic in Haverhill, Massachusetts (1926) an organism, which is identical in description with *Streptobacillus* *moniliformus*, was reported to have been present in ten out of twelve blood cultures. No serologic tests were taken in these cases, however.[5a]

Murrel states that rat bite fever, although positively associated with other organisms than the spirilla, is invariably negative in regards to false-positive reactions,[5a] whereas Davis concludes that reactions are quite frequently positive with *Streptobacillus moniliformus*.

From the above, it will be seen that no conclusions are possible, either as to exact etiology of rat bite fever, or concerning the occurrence of false-positive reactions.

Relapsing Fever: This is a very rare disease in the United States. One case has been reported in the United States, in which an undoubted relationship existed between a positive Wassermann reaction and an injection with *Spirochaeta novyi.* No evidence of syphilis was present, and the blood Wassermann returned to normal within a one month period. The infection was treated with three doses of mapharsen, and with each blood serology there was a gradual disappearance of the positive reaction. In the discussion of the above article, it was pointed out that in California there is an endemic infection of chipmunks with the spirochete of Obermeier, which is transmitted to the human being by the bite of a tiny tick and which produces relasping fever. Transient positive Wassermann reactions have been noted with this disease.[6a]

Pai has reported that seven of fourteen cases had a postitive Wassermann reaction which became negative after treatment with only small doses of arsenicals.[4]

Although in the above reports small doses of arsenicals were used for treatment of the disease, it appears that this disease rather frequently produces false-positive Wassermann reactions.

Disseminated Lupus Erythematosus: Coburn and Moore have reported 30 cases of this disease in which 11 patients had positive Wassermann reactions and 13 had positive Kline tests, without history or clinical evidence of syphilis. The Wassermann reactions fluctuated from anti-complementary to positive or negative and the Kline tests from four plus to zero, irrespective of the presence or absence of pyrexia. Eight patients had four plus or anti-complementary Wassermann reactions and all of these sera gave four plus Kline tests. Three had one plus to four plus Wassermann reactions with positive Kline tests.

Tests were performed concerning temperature increases on syphilitic and lupus serums. It was found that lupus sera, which originally gave a weaker reaction than syphilitic serum, became more strongly positive as the temperature was raised (strongest at 62° C.). This suggested that in addition to the differences in quantity of antibody there was a qualitative difference between the antibodies of syphilitic and lupus serums.

Globulin fractions, separated electrophoretically were tested for activity with the Wassermann and Kline antigens. The nitrogen content of the samples to be tested was determined and protein solutions of approximately equal nitrogen values were used. The antibody was found to be mostly in the gamma fraction and partly in the beta fraction, indicating that it migrated with a motility between that of the beta and gamma globulin.[7a]

Measles and Scarlet Fever: It is reported by Pockels that in 10 out of 134 cases of measles either positive Wassermann reactions or positive Meinicke turbidity tests were obtained. The blood was taken between the sixth and thirteenth day of the disease. On repitition of the tests several days later, a negative result was usually obtained.[13]

Another series is reported by Lane who found that in 56 cases of measles eight had strongly positive Kahn tests. These positive cases appeared within the first three weeks of the disease. Five of the cases tested at a later date were found to be negative, without receiving antisyphilitic therapy.[4]

Other authors have not reported any false biologic reaction in their studies, although it seems likely that if patients with the disease were tested at frequent intervals during the course of the disease that a more definite conclusion could be arrived at.

Scarlet fever in the earlier literature was reported as being a frequent cause of false-positive Wassermann reactions, but in the last decade there have not been any reports of cases adequately followed. In a personal communication to Davis by Lund,[4] it was stated that patients with scarlet fever had the same level of "natural Wassermann antibody" as the normal population.

Definite conclusions concerning the above diseases can only be hoped for if large series of cases are followed.

Pneumonia and Other Respiratory Infections: Shifting Wassermann reactions are reported in a series of 52 cases of a typical bronchopneumonia although no exact clinical follow-up was done in these cases, nor was the number of cases in which this occurred reported.[8a]

Mention is made in a report by Bridgeman[2] of one case of pneumonia and bronchiectasis in which false-positive reaction occurred. Another report[12] found four cases of pneumonia in which positive serology returned to negative after subsidence of acute infection.

European writers have repeatedly, in the last decade, made mention of the fact that false-positive Wassermann reactions occur with some frequency with respiratory infections.

Hegglin in 1941 reported 19 cases in adults of an atypical pneumonia with repeated strongly positive Wassermann reactions which became negative within a few weeks. He suggested a new disease entity "Wassermann positive pulmonary infection" since positive tests were found to occur uniformly in cases with this type of patchy, soft pulmonary infiltration, whereas in 680 cases of typical pneumonia and bronchopneumonia only 2.8 per cent had transient weak reactions to one or another tests. He made no attempt to relate these atypical cases to "virus" of variety "x" pneumonia.[4]

Three cases of sore throat are reported by Mohr, et al., but unfortunately two cases were not seen at the time of acute infection, and did not have tests for heterophile antibody done at this time. Both of these may have had some type of sore throat other than that associated with infectious mononucleosis.[12]

Two American reports[4] emphasize that false-positive biologic Wassermann reactions occur in a large proportion of patients with only mild infections.

It can be seen from the above that atypical pneumonia may be a frequent cause of these reactions, but no satisfactory conclusion as yet can be drawn concerning other less clearly defined respiratory infections.

Miscellaneous Infections: Lymphogranuloma inguinale has recently been reported to produce false-positive biologic reactions, with their disappearance after treatment of the disease without antisyphilitic therapy. It is suggested by Cardon[3] that hyperprotenemia, which is frequently associated with this disease, may be the factor producing these reactions.

Other diseases such as typhus, typhoid, mumps, and chicken pox are occasionally in the literature, but no definite information is available concerning these conditions. Weil's disease, Vincent's angina, diphtheria, glanders, rheumatic fever, infectious hepatitis, chancroid fall into the same category.

Four cases of acute suppurative otitis media, one case of streptococcus septicemia, one of cellulitis and furuncle, one acute appendicitis, and one paratyphoid A, have been reported to have given transient false-positive Wassermann reactions.[2]

Subacute bacterial endocarditis was frequently mentioned in earlier reports as a cause of false-positive reactions, but no recent adequate report is obtainable.

Non-Infectious Conditions: Hyperglobulinemia has been previously mentioned as a possible cause, although infrequent, of positive reactions. This condition is believed by some to be merely a reaction of an ununderlying injection process, and is not regarded as an actual cause of false-positive reactions.

One case of gastrointestinal allergy is reported as giving rise to false reactions, as is occasionally pernicious anemia, severe jaundice, and leukemia xanthomatosis.[18]

Menstruation, pregnancy, malignancy, diabetes, acetic acid poisoning, ether anesthesia, and sulfanilamide administration, have occasionally been reported but do not fit into our modern knowledge of antigen antibody reaction, and have no recent confirmation.[4]

It is to be remembered that at times excess bacterial growth in serum, excess fat and digestive products, and high barometric pressures may produce transient false, although not biologic, reactions.[18]

Spinal Fluid Wassermann: According to Davis, there is no clearer evidence today of the production of false-positive reactions in spinal fluid, although it is concluded by Scott in a controlled study that false-positive reactions occurred in seven cases of meningitis—three tuberculous, two meningococcal and two asceptic lymphocytic. He points out that false-positive reactions may occur in non-syphilitic patients during the course of pyogenic, aseptic or tuberculous meningitis, and that the diagnosis of neurosyphilis based on a positive spinal Wassermann reaction alone is unjustified in patients suffering from meningitis and other acute intracranial disorders until repeated examinations, performed after the processes have subsided, demonstrate the continued presence of reagin.[17]

APPROACH TO PROBLEM

Davis[4] points out that the most promising and rational approach to the detection of delicate differences between immunologically cross-reacting antibodies would involve immunological methods. These would include: (1) purification of the antigen obtained from mammalian tissue (beef heart); (2) testing for entirely different antibodies produced in syphilis, such as (a) antibodies of the Donath-Landsteiner type, (b) protective antibodies, and (c) antibodies reacting with spirochetes immunologically as close as possible to the true virulent *Treponema pallidum*, and (3) dissociation of antibody from floccules, on the supposition that certain false-positive serums react by virtue of physico-chemical abnormalities not involving dissociable antibodies.

The above is presented mainly to emphasize the complexity of the problem as it now exists, and to point out the definite need for future work in such a vast public health, as well as individual, problem.

METHOD OF APPROACH TO CLINICAL STUDY OF FALSE BIOLOGIC WASSERMANN REACTIONS

(1) Take careful history in reference to syphilitic infection, and do thorough physical examination, including complete dermatologic examination.

(2) Take careful history as to: (a) symptoms of some intercurrent infections of whatever nature; (b) serum treatment or immunizations; (c) vaccinations within four months preceding the questionable serologic tests.

(3) Perform a careful physical examination for evidence of acute infection preceding the questionable serologic tests, with special reference to lungs, spleen, and lymph nodes. The lymph node enlargement of infectious mononucleosis may persist for several weeks after the acute infection subsides, as also may basilar remains of an acute infection of the upper respiratory tract.

(4) A thorough search for malarial parasites, preferably by thick blood film technic, as this is a very important source of biologic false-positive Wassermann reactions.

(5) Make a blood smear examination for infectious mononucleosis. Abnormal cells of the lymphocytic series are especially important. The cytologic abnormalities of infectious mononucleosis tend to disappear promptly with subsidence of symptoms of the acute illness, and the smear may be normal at a time when the false-positive serologic tests persist.

(6) Do a heterophile antibody (Paul-Bunnell) test. This is fairly specific for infectious mononucleosis, and the antibody tends to persist in the blood for from four to five months after the onset of acute symptoms of illness and gradually disappear. The disappearance of the positive

serologic tests for syphilis does not necesarily parallel the disappearance of the heterophile antibody, and may be brief, or may persist for as long as three to four months after recovery from the illness.

(7) Determination of the sedimentation rate. This is considerably increased in early syphilis and in neurosyphilis; it may be normal or slightly increased in late or latent syphilis. It is also greatly increased in infectious processes. In the latter it will usually fall to normal as the infection subsides, whereas in syphilis will usually remain elevated.

(8) Perform various serologic tests repeatedly. Repeated strongly positive (four plus) quantitative tests are rarely found in the absence of syphilis. It has been pointed out that highly sensitive flocculation tests or precipation tests should not be used alone or as a screening procedure unless one wishes to have a harvest of non-specific reactions. When several technics are used the physician will be justified in suspecting as false positive results which are paradoxically negative, with more sensitive and positive with less sensitive technics.

Also serologic tests should be repeated in several different laboratories of known excellence in order to rule out technical error. Tests which are positive in one laboratory and negative in all others, should be suspected as false-positive.

(9) Perform Kahn verification tests, and if possible the complement fixation test with Reiter or Kazan strain *Treponema pallidum.*

(10) Test patient's serum with entirely non-specific antigens. Certain persons apparently possess the peculiar property of reacting positively to all complement fixation tests.

(11) Serologic follow-up should be prolonged; frequent quantitative serologic examinations should be done. False-positive tests tend spontaneously to fall in titer. Those due to syphilis tend to remain unchanged or to rise. Members of the patient's family should also be examnied, and sexual contacts as well, if possible.

A positive serologic test is not an emergency, except in the case of pregnancy. Although the results of treatment are best in early cases, delay of a few months in a case of latent syphilis is not likely to affect the outcome appreciably. Three months is a reasonable deadline for false tests to return to normal, and may be used by the civilian practitioner before antisyphilitic therapy is begun.

(12) Cerebrospinal fluid may be examined if a decision cannot be reached. When there is genuine reason for believing that the positive result in false, do not adopt spinal puncture as the first step in the investigation, since often a clear cut explanation can be found for the supposed false-positive result. Unless the reason for false-positivity is obvious, however, the patient should not be dismissed without a study of the spinal fluid, lest asymptomatic neurosyphilis be missed, with subsequent tragic results.

(13) Always suspect false-positive result if: (1) tests are weakly positive or doubtful; (2) a conflict between precipitation tests and complement fixation tests; (3) a repetition is positive, but titer remains low or is extremely fluctuant, (4) or if there is a decline of a titer to negative on weekly tests from one to three months without treatment.

CONCLUSIONS

Biologic false-positive serologic tests for syphilis are of definite interest to the physician. Numerous conditions have been reported to cause such reactions, although at present there is no satisfactory explanation for the occurrence, nor is there any exact agreement on the percentage of these reactions in various diseases. Nevertheless, the conditions mentioned above must be in the mind of physicians, so as to avoid an erroneous diagnosis of syphilis, and to place on his patient a tag which is of so great a social, as well as psychologic, trauma.

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BED REST IN HEART DISEASE[*]

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Without a doubt, the most commonly used therapeutic principle in modern medicine today, is bed rest. It is used by the layman as well as the physician as the fundamental basis in the treatment of anything from a minor to a major pathologic condition. In spite of its common usage, there has been very little scientific investigation of its effects and the extent to which the period of inactivity should be continued depends on individual subjective medical opinions, which are for the most part, empirical.

Hoff[15] stated in 1944, "the re-examination of established principles often proves to be as effective a means of progress as explanation of new fields. This is especially likely to occur in those instances where the weight of long tradition or great authority and the superficial appearance of reasonableness have combined to discourage the critical analysis of a doctrine."

Because of the urgent need for hospital space and nursing care in the last few years, the problem of bed rest has been forced on the medical profession. It first received the attention of the surgeons and obstetricians who found that the length of bed rest could be diminished markedly without any apparent harm to their results. This has caused it to become a much discussed problem in every field of medicine including cardiology. Although bed rest is considered by most present day authors as one of the most fundamental principles in the treatment of heart disease, this has not always been the case. In fact, its value was not even mentioned until the latter part of the nineteenth century.

HISTORICAL REVIEW

Most of the authorities on heart disease in the nineteenth century believed that the cardiac muscle reacted in the same manner as skeletal muscle to exercise or rest.[7,27] Thus, they thought that the logical therapy for myocardial insufficiency was exercise, which would strengthen the cardiac muscle and thereby produce a heart which was better able to supply its demands. By the same reasoning, rest would cause fatty degeneration of the heart muscle and thus decrease its ability to do its work. In 1945, Stokes[7] warned the profession against this latter danger and stated that any patient with heart disease must learn to give up the luxurious habits of life.

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Exercise was used even in cases of severe failure or acute myocarditis. In 1876 Schroetter[27] said in speaking of acute myocarditis that the chief indication would naturally be, as in inflammation of any other organ, to put the patient in a complete state of rest, but such a measure would be dangerous as it "would only tend to increase the great source of danger in the disease, viz., weakness of the heart."

Austin Flint[7] wrote, in 1886, that patients with heart disease exchanging the habits of activity for complete rest are likely to become rapidly worse.

In the eighties, the Pertel treatment of graduated periods of exercise in the form of mountain climbing became exceedingly popular and was used by nearly every authority on heart disease. The Nauheim baths introduced a few years later were based on similar principles and consisted of carbon dioxide steam baths with passive massage followed by graduated periods of exercise.

Davis[7] states that the first comment on the bad effects of exercise in heart disease was made by Corvisart in 1812, and credits Hope (1839) and Bamberger (1857), as being the first to emphasize the importance of rest. Pratt,[27] however, could find no writer prior to 1872 who recognized the value of rest, and credits its introduction to Fothergill, Gibson and Balfour. In 1872 Fothergill published the first edition of his book on "The Heart and Its Diseases" in which he states that "perfect rest, especially in bed for a time, combined with proper remedies, will give relief in every case of mere failure of the muscular structure of the heart, not complicated by valvular disease."

Gibson[27] presented an article in 1877 comparing the effects of rest and exercise in rheumatic fever. His cases covered a period of 26 years and clearly showed the valve of rest on the heart. However, he did not seem to recognize the great significance of his findings as far as the heart was concerned and it remained for Fothergill to do this in the second edition of his book in 1878. In spite of this Pratt[27] states that Balfour's book which appeared in 1876 indicated that he had a much clearer conception of the value of rest, and he may have been the first really to teach and emphasize this point. Balfour[27] says "You must never forget to insist upon rest as the most important part of the treatment of heart disease, a rest which must be more perfect and complete, the more serious the cardiac disturbance is." The true significance of this statement can only be realized if one keeps in mind that it was made at a time when most men were advocating exercise as a treatment of heart disease and warning against the dangers of rest.

Broadbent,[7] in 1898 was the first to state a definite period which bed rest should be continued. He said, "Rest is in many cases an important part of the treatment and when the state of the patient demands it, rest in bed is of the greatest service; but one of the questions calling for the greatest exercise of judgment is to decide when absolute rest is necessary. There are circumstances in which a month or six weeks in bed will prolong life for as many years. Even when severe symptoms are present, to insist upon confinement indoors is sometimes to add to the suffering of the last few months of life without adding to its duration." This same question is one aspect of the problem of bed rest applied today. In spite of his belief in bed rest in some cases, Broadbent continued to advocate the Pertel treatment in the majority of cases. Pratt[27] believes Sir William Osler was the first to write about the value of rest in this country.

He wrote "Disturbed compensation may be completely restored by rest of the body."

During the next few years most of the authorities such as Brunton, Osharner, Mackenzie and Helbut began to recognize the value of bed rest, especially during the acute stages of failure, but continued to stress the importance of exercise during convalescence. Brunton[7] advised his patients that he "would not take one beat out of the heart that could possibly be avoided." Mackenzie[7] wrote, "when exhaustion has gone so far that breathlessness or pain is easily induced and objective signs, such as dropsy, are present, rest is imperative. While other remedies may be necessary, rest is the most potent remedy of all. When the patient is fit to go about, he should be encouraged to take exercise in the fresh air. The amount of exercise he may take will depend upon his strength and this strength may be gauged by his sensations. So long as a man who is taking exercise remains free from distress or discomfort, then exercise is doing him good."

Pratt[27] in 1920 introduced the idea that it may be possible to build up the cardiac reserve further after all the signs and symptoms of failure had disappeared by keeping the patient in bed for a longer time. He also used initial and prophylactic periods of bed rest in the treatment of angina pectoris. He discredited the logic behind the value of exercise in the treatment of heart disease on the basis of three general facts; first, that cardiac muscle is a distinct type of tissue with special functions and structure, different from skeletal muscle; secondly, that the comparison is of a healthy muscle reaction to exercise to that of a diseased muscle; and thirdly, that no amount of exercise could possibly change valvular deformities, reduce blood pressure or decrease the amount of coronary sclerosis, the factors involved in most cases of cardiac disease.

PRESENT CONCEPT OF BED REST

At the present time, bed rest is generally accepted as one of the most important parts of the treatment of heart disease. However, that is as far as the agreement goes. How long should the patient be put to bed? What amount of activity should be allowed in connection with bed rest? What position in bed should the patient be allowed to assume? Should the patient be allowed to sit up in a chair? These are a few of the questions for which there are as many answers as there are men writing on the subject.

In active rheumatic heart disease there is probably more agreement as far as bed rest is concerned than in any other clinical form of heart disease. As pointed out earlier in this paper, this was probably the first type of heart disease in which bed rest was proved of value in both the prevention and cure. Paul White[41] states, "In acute rheumatic heart disease, the most important therapy is complete rest in bed. The patient should not be allowed out of bed until the signs of acute infection have completely subsided, even if it takes months or a year or two." This same view is taken by nearly every authority. Sprague[30] gives the signs upon which the decision concerning the lack of activity of the disease are concerned as:

1. Absence of fever for two weeks.
2. Normal white blood count and sedimentation rate.
3. Evidence of congestive failure gone.
4. P-R interval normal.
5. No subcutaneous nodules.
6. No chorea.
7. The patient has no respiratory infection within three weeks which might serve as a reactivating episode. Levine[17] points out that this condition may persist showing only slight evidence of activity for a long period of time. In such cases the question arises whether the decrease in heart damage will be in proportion to the length of bed rest. In such cases social, economic and other factors unrelated to the disease condition itself, must enter into one's decision concerning prolonged bed rest.

It is important to realize that because of the relative youth of these patients, many of the disadvantages and dangers of prolonged bed rest which will be discussed later in this paper apply to only a small percentage of this group. For this reason acute rheumatic heart disease has received little attention in the recent discussions concerning bed rest in heart disease. The problems of bed rest inactivities in bacterial endocarditis are practically the same as those for active rheumatic heart disease. Likewise the agreement is the same that absolute bed rest should be maintained until all signs of activity are gone. Those conditions given by Sprague as signs of lack of activity in rheumatic fever may also be applied to this condition. Of course, in addition to those signs the blood cultures should be negative.

Bed rest has been used as the treatment of some cases of angina pectoris. Hoyle and Evans[16] reported a series of 23 average cases of angina pectoris which they treated with a period of one month or more of complete bed rest. Seventeen of their cases showed subsequent improvement, seven showing great and ten showing slight improvement. The effect seemed to last for four to nine months following return to the pre-rest activities and decreased the severity of the attacks more than the frequency. From the results, they recommended a period of complete bed rest for two to six weeks in patients who have not improved or are becoming worse in spite of treatment. They state that those patients with pain of recent onset, and those with hypertension are most likely to benefit from this.

Wayne and Laplace[38] conducted a careful study on the effect of rest and various drugs on five cases of angina pectoris and "were surprised that the improvement was so slight following rest." Two patients whose condition was of recent onset benefitted most.

Of interest along this same line, are the findings of Harrison.[14] He found that 14.2 per cent of his patients with angina pectoris gave a history of anginal attacks occurring while they were resting in the recumbent position, whereas none reported the onset of pain to occur while resting in the sitting position. Electrocardiographic tracings taken during an anginal attack brought on by recumbency showed typical changes of angina pectoris. These findings would seem to indicate that the coronary blood supply is lower in proportion to the demand in the recumbent position than it is in the sitting or upright position. Harrison[13] concludes that rest in bed for more than a day or two has no place in the treatment of angina pectoris except in those patients who are especially liable to develop in the immediate future myocardial infarction, as indicated by increasingly frequent and prolonged attacks at rest.

In the acute stage of congestive heart failure, nearly everyone believes that absolute rest in the position of choice is essential. If the dyspnea is severe it would be practically useless for anyone to insist on anything besides absolute rest because the symptoms themselves limit the amount of activity possible. These same symptoms force practically every patient to remain in the sitting position whether it is in bed or in a chair. Once the acute attack has begun to subside, the problem is the same as that encountered in cases of mild or chronic congestive failure.

It is in those cases of mild or chronic congestive failure where almost every writer has his own "pet" theories concerning bed rest.

Patrick[24] states that rest in bed is the most important part of treatment in cardiac failure. He claims that getting out of bed to sit up for breathlessness only strains the heart further and increases the dyspnea. Therefore, one should keep a patient confined to bed in the recumbent position, even though one may have to tide him over the difficult first stage with large doses of morphine and opium. After improvement takes place and a few weeks of complete rest, the patient may be allowed up a little at a time, but for months he should spend much of his time in bed.

Davis[7] has reported one of the very few series of cases in which the effects of prolonged bed rest is compared with the effects of the more usual periods of rest. He had eleven patients who were treated with absolute bed rest for three to twelve months, according to the severity of the failure, after all symptoms had disappeared. Following this period of initial prolonged rest, they were warned to reduce their activities below the level of activity performed before the onset of failure and also to take regular periods of prophylactic bed rest. Seven out of the 11 cases were free from evidence of congestive heart failure three or more years, and three had maintained that good health for a period of five or more years up to the time of the report. These cases were compared with 50 controlled cases treated for an average of two weeks in the hospital and then discharged with the recommendation to rest some more at home. Thirty of these patients out of 40 cases having two or more hospital admissions, returned in congestive failure within one year after the initial attack. Of these, 21 returned within six months, 15 within three months and several within two or four weeks.

From these results he concluded that many patients at the time of discharge from the hospital are in a condition of comparative failure, in spite of the absence of the usual signs. He outlined a general program which he believes should be used in the treatment of congestive failure based on the duration of failure. It is as follows:

Duration of Cardiac Failure	Therapeutic Minimum	Bed Rest Desirable	Prophylatic Bed Rest With Bathroom & Table Privileges
2 or less weeks	12 weeks	16 weeks	1 week out of 3
4 or less weeks	16 weeks	24 weeks	1 week out of 6
8 or less weeks	32 weeks	48 weeks	1 week out of 4
12 or less weeks	48 weeks	72 weeks	1 week out of 3

Strauss[31] on the other hand believes that absolute rest is rarely required or desirable except in the acute or terminal stages. He points out that absolute rest with mental perturbation may, and usually does, result in greater strain on the circulation than mild physical activity and peace of mind.

Eggleston[10] allows his patients to assume any position they prefer which is usually the sitting position to begin with. Then as dyspnea decreases he has them gradually assume a more recumbent position. They are confined to bed for at least one week after all evidences of heart failure have disappeared. He recommends a prolonged convalescence with gradually increasing activity over a period of time adequate to permit the damaged heart to regain the maximum of reserve power still left to it.

Although Powers[26] believes that absolute rest is necessary in treating a severe grade of insufficiency, he believes it should be in the position in which the patient is most comfortable. He points out that the recumbent position is not necessarily a restful one so far as the heart is concerned.

Levine[17,18,19] has been one of the foremost authorities in the warning against the dangers of recumbency in the treatment of congestive failure. He[18] believes that he has occasionally saved a patient's life by taking him out of bed and placing him in a chair. He warns that although recumbency may reduce the heart rate and blood pressure, the increase in venous flow and resulting increase in pulmonary congestion more than cancels out these effects, and the result is an increase rather than a decrease in cardiac work. Many physicians believe the disappearance of ankle edema is proof of the value of bed rest. In relation to this, Levine[19] states, "It must be borne in mind that fluid in the legs may be unsightly but does comparatively little harm, whereas fluid in the lungs is a dangerous handicap." He does not make any claim that rest is not beneficial for cases of heart failure, but he questions whether the recumbent position is the position of greatest rest. He also emphasizes the dangers of pulmonary embolism which result from prolonged bed rest. He concludes that some severely ill cardiac patients, especially those who experience paroxysmal nocturnal dyspnea should not be kept flat in bed until active cardiac treatment has been well advanced in improving the circulation. During the early days they may sit in a chair and should be urged to exercise their legs or take short walks in their room several times daily. He keeps his patients on semi-complete bed rest for one week following the disappearance of symptoms and advocates a period of gradual convalescence with reduction of activities following this. Because of the great dangers associated with bed rest, especially if prolonged, Harrison[13] has recently stated that persons with congestive heart failure should be allowed out of bed for several hours a day as soon as severe dyspnea at rest has subsided. The other form of heart disease, of which there is a great amount of controversy as to the optimum position and the amount and degree of bed rest desirable, is coronary occlusion. As with congestive failure, most authorities agree on the treatment of the acute phase. Thus, these patients are all put on absolute bed rest which is usually in the recumbent position because of the associated shock. The other factor on which most authorities agree is that these cases must be highly individualized[44] and treated accordingly. In spite of this there are some general conclusions which most clinicians arrive at and these will be briefly given.

BED REST IN CORONARY OCCLUSION

Master[21] believes that these patients should be put on complete and absolute bed rest for a minimal period of six weeks. During this period they are not allowed to do anything for themselves which requires activity. Although he does not state that the patient should be kept in the recumbent position, most writers imply this unless specifically mentioned otherwise. This period of absolute rest is followed by a period of gradual convalescence.

In an admittedly very unscientific manner, Bradley[6] concluded that every case of coronary occlusion, no matter how severe, should have a minimum of three months of complete and absolute bed rest. He placed his patients in a bed with the head of the mattress thirty to forty inches above the foot. He believes that granting bathroom privileges tends to give the patient a false idea concerning his condition and he becomes more lax in the amount of bed rest. These conclusions were severely criticized by W. C. Blake in discussing the paper. He pointed out that no minimum could be set for all cases, but that the usual period of complete bed rest in uncomplicated cases was four to eight weeks followed by a period of gradual convalescence of six to eight weeks.

Sprague[30] also emphasizes that the period of confinement to bed is variable. He judges the progress of his patients by the temperature, pulse, blood pressure, electrocardiogram, white blood count, sedimentation rate, absence of gallop rhythm and cardiac failure and freedom from pain. He gives the shortest period of complete bed rest as three weeks, the average as six weeks, and the maximum as several months. If his patients have trouble with the use of the bed pan, they are allowed to use a bedside commode. He emphasizes that as soon as it can be tolerated, the patient should be encouraged to move his legs and feet around in order to help prevent the occurrence of pulmonary embolism.

S. M. White[42] believes that at least eight weeks of complete bed rest (position not stated) are necessary in coronary occlusion "if one is to minimize the likelihood of such crippling sequelae as aneurysm of the heart wall with its attendent rupture, intra-aneurysmal thrombosis and general interference with good function of the remaining muscle." This requirement is a minimum and is increased if there is recurring pain, signs indicating extension of the infarcted area, persistent low blood pressure or any other features suggesting delay in the firm fibrous replacement desired.

Paul D. White[41] states that most patients who die because of a coronary occlusion, do so in the first few days. A small percentage died during the second week and a very few died in the rest of the first year. He gives ventricular filbrillation as the most common cause of death in these cases, although rupture and congestive failure do kill a few. He recommends bed rest for a minimum of one month following occlusion, the first two weeks of which should be absolute. In the second month the patient should gradually be given more activity by sitting up some the first week, walking a little the second week, climbing a few stairs the third week and taking leisurely strolls outdoors the fourth week. It is preferable to have one more month of reduced activity if possible.

There has recently been a growing tendency among many physicians, especially Harrison and Dock, to decrease the period of initial bed rest and also to question whether the recumbent position is the position of choice.

Harrison[13] points out two examples of myocardial infarction not treated by prolonged bed rest which are of special interest to the medical profession, namely the cases of John Hunter and Sir James Mackenzie. In 1773, John Hunter at the age of 45 years had what may possibly have been a myocardial infarction. In 1785 he began to have a series of anginal attacks and within a few weeks experienced what was undoubtedly a true myocardial infarction. From that time until his sudden death in 1793, he had numerous similar seizures induced by exercise or emotion and at his autopsy marked coronary sclerosis and two large old infarct scars were found. Although he apparently never spent any longer period in bed, he continued to be quite active during his last years and accomplished much of his best work during this time. In spite of this, he lived at least eight years and possibly twenty years after the onset of symptoms indicating serious heart disease.

Sir James Mackenzie first began to experience anginal attacks in 1907 which became more severe and probably represented infarction in 1908. He died in 1925 and during that time had several attacks of typical infarction. Autopsy showed several areas of infarction of the myocardium and marked coronary sclerosis. As far as Harrison was aware Mackenzie never went to bed for more than a few days at a time and continued to be active even to the extent of playing golf until 1923, when he was forced to quit because of exercise intolerance.

Is one to assume that these two men would have lived any longer or been any happier if they had been treated with prolonged bed rest? Although Harrison is forced to admit that no conclusions can be drawn from two isolated cases, he believed that these examples should at least lead one to adopt an open mind concerning the questionable value of prolonged bed rest.

After considering the advantages and disadvantages of prolonged bed rest Harrison[13] concluded, "Following myocardial infarction, recumbency should not be prescribed for a longer period than two to three weeks after the more acute and alarming symptoms have subsided. The recumbent position should not be enforced on patients who are more comfortable sitting. Other things being equal, it would appear wise to allow elderly patients out of bed sooner than younger ones." Dock[8,9] describes many of the dangers of prolonged bed rest which will be discussed later in this paper and arrives at much the same conclusions as Harrison.

Levine[17] states that he is inclined to endorse this view in the mild cases and prescribes greater movement of the legs in all cases than was the custom formerly. He states, "One of the most common and harmful errors in the management of acute coronary thrombosis is to outline lengthy periods of invalidism and convalescence. It generally will be found that the degree of pain will be no greater if the patient is ambulatory. What is more aimless, is to advise restricted activities, as is frequently done, confinement in bed, merely because of weakness. After the first two months, such weakness will not be helped by a program of inactivity. The entire illness, coupled with the alarming precautions taken by the physician and family, produces a state of fear, which often results in a profound neurasthenia and depression. Considerable weakness is necessarily part of the illness itself. This is further accentuated by the immobility during the early weeks in bed. To this is then added the neurasthenic state that finally may condemn the individual to a permanent useless existence."

Wolferth[14] states that one sees patients with acute coronary occlusion who continue their ordinary activities without interruption, some because they disregarded medical advice and others because the true nature of their illness had not been recognized in its early stages. Although these are obviously the comparatively mild cases, many of them have done well, in fact, sometimes much better than after their condition was recognized and they were placed in bed.

In order better to understand the effect which bed rest has on the heart, circulation, lungs and body in general, a review of some of the physiologic principles involved is indicated. It is admitted that all of the physiologic effects have not been sufficiently investigated at the present time. In fact, most of these changes are fairly well established and have been worked out on normal subjects and therefore do not necessarily apply to pathologic conditions. However, it is believed that there is sufficient material for one to derive certain general conclusions as far as the clinical effect of bed rest in heart disease is concerned.

The principle upon which the use of rest in the treatment of heart disease is based, is the finding that the metabolic rate is decreased when the body becomes less active, which in turn results in a decrease of the work required by the heart. Since it has been shown that most organs heal more rapidly when put at rest, this is also assumed to apply to various heart conditions. As has been previously stated, nearly every present day authority agrees that rest is one of the most valuable principles in the treatment of heart disease. The problem left before us then is in what position, sitting or recumbent, is the work of the heart decreased the most? A few of the various factors which may be altered by a change in the position of the body will be discussed.

A decrease in cardiac output is generally accepted[1,3] as the fundamental change which occurs in the production of congestive heart failure. This may be caused by rapid heart action, auricular fibrillation or weakness of the myocardium. According to Best and Taylor,[3] the cardiac minute volume output is greater in the recumbent than in the erect position. This would tend, in the normal individual, to equalize any increase in venous blood flow which occurs in the recumbent position. However, Altschule[1] states that besides showing a decrease in cardiac output, patients with congestive heart failure fail to exhibit the normal response of increasing output in proportion to the rise of venous pressure or an increase in work. Thus one of the factors becomes apparent why the recumbent position often causes an increase in pulmonary congestion in persons suffering from heart failure. Altschule[1] also states that the work of the heart is directly proportional to its output. Therefore, if the diseased heart does show any increase in cardiac output upon lying down it would tend to increase the work of the heart.

Another factor which has been found to show marked change in different positions, is the blood volume. Thompson, Thompson and Dailey[34] found that in the standing position there was a loss of approximately protein-free fluid from the blood. This amounted to 11 per cent of the total plasma volume and thus resulted in a relative hemoconcentration. These findings have since been confirmed by Waterfield[37] and Perera and Berliner.[25] However, the significance of this change becomes clearer if we turn the statement around as Perera and Berliner did. Thus, a hemodilution occurs when a person changes from the standing to the recumbent position which amounts to an increase of blood volume of 10 to 15 per cent. Although it is probably not as great, a proportional change must occur when the patient changes from the sitting to the recumbent position. Perera and Berliner[25] state, "The effect of the horizontal position upon the resting individual is comparable to the administration of a slow, but sustained infusion. It seems probable, therefore, that in patients already subjected to left ventricular strain by virtue of underlying organic disease, the increase in circulating plasma volume accompanying rest in the horizontal position may prove a significant factor in inducing paroxysmal dyspnea and pulmonary edema."

Altschule[1] claims that the total blood volume is increased in all cases of congestive failure. The red blood count and hemoglobin show a corresponding increase so this may be an attempt of the body to compensate for the relative anoxemia which occurs. In any event it would seem that the increase in blood volume would further add to the work required by the heart.

Sewall[29] found that in changing from the recumbent to the standing position there is always a rise in diastolic pressure and a fall in pulse pressure. The systolic pressure does not change materially or show a slight rise. This would cause an increase in the force against which the ventricles must work in the sitting position. Dock[9] however, has claimed this change is negligible as far as the work of the heart is concerned. From another point of view, if one remembers that the coronary flow is directly proportional to the diastolic pressure,[2] this increase in diastolic pressure may actually be beneficial in cases of coronary disease.

The pulse rate increases about twelve to fourteen beats per minute when one changes from the recumbent to the upright position according to Sewall.[29] Field and Bock[11] reported an average of only two beats per minute increase in changing from the recumbent to the sitting position. Although this would tend to increase the work of the heart, there is so little change that it is doubtful if it has any effect. Even if the change were greater, it would cause a decrease in the stroke volume at the same time, which would seem to be the important factor in the production of cardiac aneurysm or rupture following infarction.

Thompson et al.[33] and Field and Bock[11] showed the position of the body to have a direct relationship to the rate of venous flow. Field and Bock state that the venous flow in the recumbent position was 33 per cent greater than in the sitting position. Perera and Berliner[25] showed an increase in venous pressure at the auricles and in the veins of the head when a person changes from the upright to the recumbent position. Altschule[1] states that the increase in venous pressure in the respiratory centers is one of the factors causing dyspnea in the recumbent position.

McMichael and McGibbon[23] have demonstrated that in normal persons, there is a decrease in the total lung volume of over three hundred cubic centimeters and a decrease in the vital capacity of about two hundred cubic centimeters in the recumbent position. Many authorities have found a decrease in vital capacity in patients with congestive heart failure and Levine[18] believes that the difference between the recumbent and sitting position in these patients is even more marked than that which occurs in normal persons. These changes have been ascribed by Altschulte[1] as due to; (1) less effective pulmonary ventilation in the recumbent position due to shift of the diaphragm upwards and the limiting of thoracic motion; (2) changes in the amount of blood in the thorax as a result of increased pulmonary congestion; and (3) decrease in the negative intrapleural pressure.

The last factor, but far from the least, is the effect of the position on the basal metabolism rate. Best and Taylor[3] state that there is little or no difference in the basal metabolic rate taken in the sitting or recumbent position.

Although it must be reemphasized that most of the above findings were a result of study of normal persons, their significance seems great enough to allow one to conclude that the sitting position is at least as restful to the heart, if not more so, than the recumbent position. Therefore, there seems to be no indications from a physiologic standpoint for the physician to insist on the recumbent position in the treatment of heart disease, except perhaps in the early phase of myocardial infarction when there is danger of cerebral anoxia due to the shock.

There has been very little work done along experimental lines on the value of rest in the treatment of heart disease. The only two studies found by the writer were on the effect of rest following myocardial infarction and they will be summarized below.

Sutter and Davis,[28] in 1931, tied off the anterior descending branch of the left coronary artery in six dogs. One dog was given a rest period of six days following which he was forced to run an hour every third or fourth day on a treadmill at a rate of approximately three miles per hour. The other dogs were forced to start exercise on the second or third day, some doing the same amount of exercise as the first dog and others exercising as much as every other day. They were autopsied at various lengths of time and the first dog was found to have a small, well contracted scar without thinning of the ventricle whereas the others had thin scars with aneurysmal bulging. Thus it was concluded that rest was helpful in producing better healing of the myocardium following infarction and early exercise tended to cause aneurysms.

While it is admitted that these findings show a definite trend, it is obvious that the number of subjects was insufficient to derive any definite conclusions. Also it is difficult for the writer to see any significance in their findings as far as clinical medicine is concerned. It is very doubtful if there is any physician who advocates running for one hour three days, three months or three years after an attack of coronary occlusion.

Thomas and Harrison[32] conducted some experiments on rats, in which the survival rate was studied in rats after damage was inflicted on the myocardium by burning the left ventricle. In rats whose activities were restricted to approximately 10 per cent of that of unrestricted controls, only seven survived out of 49, whereas 21 out of 47 survived in the control group. Unconfirmed rats resumed normal activity in five days as shown by their optional use of a treadmill in the cage. Forced, strenuous exercise (swimming) failed to influence mortality if it were not begun before 48 hours after cardiac trauma. From these experiments the authors concluded that physical activity within the range of tolerance may be more beneficial than complete rest. They call attention however, to the obvious fact that restraint, even though it may reduce muscular activity, can hardly be termed rest, and it is highly uncertain that the cardiovascular load under these circumstances is in any measure diminished.

Mallory, White and Salcido-Salgar[20] did some very comprehensive work on the speed of healing of the human heart following myocardial infarction. They reviewed the pathological findings in seventy-two cases of infarction in which the age of the infarct was definitely known. They found that the speed of healing is, in part, dependent on the size and position of the infarct and in part on the state of the remaining myocardial circulation, although the latter is of the least importance. Rupture of the heart was most common during the first week, occasionally occurred during the second week and was extremely weak. Small infarcts were almost completely healed in five weeks and large infarcts showed no discernable changes after eight weeks. On the basis of these findings they recommended bed rest for four weeks, only the first two of which need be absolute, followed by a period of gradual convalescence as already given by White.

The reasons why many authorities have recently advocated shorter periods of bed rest in patients with heart disease, are the marked dangers which accompany prolonged bed rest. Tillett[35] states, "The resulting total activity from bed rest created through disease, damage to functions that are otherwise normal, while at the same time permitting the damaged tissues to undergo repairs."

Dock[9] has especially emphasized the dangers of bed rest. He believes that "absolute bed rest kills more patients than anesthesia and all the drugs in the pharmacopoeia added together." He points out that man is the only mammal that lies on his back (a highly unphysiologic position) when he is ill.

Gastrointestinal changes are some of the most common problems encountered by the physician when he puts a patient to bed even for a short time. A high percentage of patients confined to their beds become constipated or distended and require regular cathartics or enemas. Many acquire cathartic habituation at this time which remains a problem to them even after they are allowed up. Although this does not in itself constitute a danger, it is obviously not desirable. Associated with this same problem is that of the bed pan. Many authorities advocate complete use of the bed pan, whereas others believe that the straining involved in its use may be more dangerous than if the patient were allowed to get up and use a bedside commode. This question is especially significant in cases of myocardial infarction. Dock[8, 9] believes that the main danger in using the bed pan in either position is that of repeating the Valsalva experiment. Valsalva found that whenever one takes a deep breath, closes the glottis and tightens the thoracic and abdominal muscles, there follows a series of extreme fluctuations in thoracic pressure, cardiac output, blood pressure and vagal and vasomotor tone which results in a tremendous strain on the heart and circulation. Dock[9] states that after myocardial infarction, sudden death is far more likely to occur when the patient performs this effort than from any other cause, and it is a common incitant of pulmonary embolism. Unless previously warned, the patient may reproduce this experiment on the bed pan, or when trying to push himself higher on the pillows or even when passing flatus.

Urinary retention or difficulty in voiding in the recumbent position is another problem commonly encountered in (bed) patients. These patients must then be either gotten up or catheterized, with all its well-known dangers which must be resorted to. In elderly men, bed rest often precipitates severe symptoms of prostatism. It has also been found that immobility causes an increase in the rate of calcium excretion in the urine which may in turn increase the incidence of renal stones.[9]

Prolonged inactivity also causes a decrease in the vasomotor tone and atrophy of the muscles. Backache and muscle cramps often appear as a result of a long period of confinement to bed. Bed sores were probably one of the first dangers recognized as a result of bed rest. Since cardiac patients are commonly elderly and heavily sedated, this complication is especially prone to occur, unless special precautions are taken.

Pulmonary complications are probably the most important of any of the dangers of prolonged bed rest. Hypostatic pneumonia has long been recognzied as a common cause of death in chronic diseases. This is especially apt to occur in cases of cardiac failure where the lungs already show an increase in the amount of congestion. Williams and Rainey,[43] in autopsies on a large series of patients dying with congestive failure found pneumonia to be the chief cause of death in 14 per cent, and a supplemental cause in 45 per cent of the cases. The danger of the recumbent position causing a further increase in pulmonary congestion as cited by Levine[17, 18, 19] has already been discussed. Pulmonary embolism has for a long time been considered by clinicians as more or less a surgical or obstetrical complication occurring during postoperative periods. Emphasis of the condition and more widespread recognition has shown it to be common in medical cases as well. White[40] found in reviewing 370 cases of pulmonary embolism which came to autopsy that 40 per cent were postoperative, with 58 per cent infarcts, 30 per cent were cardiac with 90 per cent infarcts and the other 30 per cent were non-cardiac medical with 62 per cent infarcts. He ascribed the high degree of infarction in cardiac cases as due to the fact that the lungs of these patients were already congested and the collateral circulation was thus at a minimum.

Williams and Rainey,[43] in their series of cases of patients dying of congestive heart failure, found pulmonary embolism to be the chief cause of death in 17 per cent and a supplemental cause in 32 per cent.

Dock[9] reports that in 300 consecutive autopsies in a New York hospital, 8 per cent were found to have died as a result of pulmonary embolism. He quotes Weststahl of a San Francisco hospital as reporting 13 per cent of patients autopsied showed signs of pulmonary embolism and it was judged to be the chief cause of death in 3.5 per cent of the total cases. He states that Swiss and German pathologists have found, by complete dissection of leg veins at autopsy, that 30 per cent of all hospital cases between seventeen and ninety years of age to have thrombi in the veins of the calf and the incidence rose to 60 per cent of all adults confined to bed for more than two weeks. The incidence rises with age, doubling at forty years, and doubling again at sixty years. Dock points out that most of these thrombi are formed in the smaller muscles of the calf and thigh as a result of pressure on these parts against the bed. Thus, they are especially common in patients under extreme sedation or in very obese patients who "lie like logs". In order to avoid this danger, he advocates a reduction of bed rest to the minimum essential and repeated movements of the legs and arms in those persons who are forced to remain in bed.

White[40] emphasizes that pulmonary embolism often closely simulates acute left ventricular failure by precipitating pulmonary edema or asthmatic breathing. It is also often difficult to differentiate from myocardial infarction. The importance of making the correct diagnosis is obvious.

Thomas and Harrison[32] believe that the incidence of ventricular fibrillation, cardiac rupture, circulatory collapse, systemic embolism and ventricular aneurysm following coronary occlusion is probably decreased by prolonged bed rest. They point out, however, that there is no evidence to indicate that these complications are more common in the sitting position than in the recumbent position. They further believe that the important factors which may be increased by prolonged bed rest are further myocardial infarction, pulmonary edema, pulmonary embolism, pneumonia and cerebral thrombosis. It is important to remember that most of those complications which are supposedly caused by bed rest occur during the first two weeks if they are going to occur at all.

The last danger which will be discussed is that of the psychologic effects of prolonged bed rest. After the individual has recovered from the acute stage of either congestive failure or myocardial infarction, there is an ever increasing desire to be out of bed. If the physician continues to urge complete bed rest for a prolonged period it would seem logical for the patient to begin to exaggerate to himself the seriousness of his physical condition. This sequence of events, all too frequently leads to a cardiac anxiety state. Harrison[13] states, "Even if we could be certain that such excessive caution would prolong life in an occasional patient, and there is no grave danger concerning this it still remains the question as to whether the objective so achieved merits the unhappiness produced in a larger group of patients by rigid restrictions."

From the foregoing presentation of bed rest and how it is used in the treatment of heart disease, it becomes obvious that one of our greatest needs in medicine today is some scientific investigation of bed rest and its various effects on the diseased human body. In spite of this, it would seem justifiable to make certain conclusions on the basis of our present clinical and experimental evidence.

1. Patients with active rheumatic fever, whether they show signs of heart disease or not should be kept on complete bed rest until at least two weeks after all signs of activity have disappeared. Rarely, in chronically active cases of long duration, the disadvantages of bed rest may outweigh the advantages.

2. Bacterial endocarditis also calls for complete bed rest until all signs of activity have been absent at least two, and perhaps a few more weeks.

3. Prolonged bed rest probably has little or no value in the treatment of angina pectoris. A possible exception to this is repeated frequent attacks of angina which may possibly be a prelude to coronary occlusion.

4. Patients in the acute stage of congestive failure should be placed at complete and absolute bed rest in the position of their choice which is almost invariably the sitting position. They should, however, be advised to move their legs frequently.

5. The physician should not be in too great a hurry to place the patient with chronic congestive failure in the recumbent position. Many of these patients may be treated just as well, during the early days, if not better, by being placed in a chair. After a certain degree of compensation has been attained, they may gradually begin to spend more time in the recumbent position.

They should be urged to exercise their legs or take short walks in their rooms several times daily. They should be kept on this semi-complete bed rest until one week after all symptoms have disappeared when they may begin a period of gradually increased activity.

6. The value of prolonged bed rest in the treatment of myocardial infarction has probably been overemphasized. Complete and absolute bed rest in the recumbent position is indicated during the initial phase of shock. Following this, bed rest in the position of choice should be continued for three or four weeks. During this period the patient should be instructed to exercise his legs regularly and may be allowed to use a bedside commode if the use of a bed pan proves to be difficult. It is very important to warn the patient against reproducing Valsalva's experiment. The use of sedatives should be kept to a minimum. Following this initial period of bed rest, a period of convalescence with gradually increasing activity extending over four to six weeks may be begun.

7. Lastly, it should be emphasized that every case of heart disease is an individual problem and the general form of therapy should be varied accordingly.

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CHECK AND DOUBLE CHECK

A statement appears in this issue of the Journal concerning a pamphlet which has been put out by the Public Relations Bureau of the Medical Society of the State of New York. This is prepared by a school teacher of Portland, Maine, Mr. J. W. Walch. We would like to comment on this pamphlet a bit in detail because we think it might be of great value to the physician to have in his office to give to his thinking patients. The pamphlet is prepared for lay groups as well as for doctors and can be readily understood by persons not necessarily physicians.

Mr. Walch asks some 133 questions, and then proceeds to answer them. From his point of view, as a layman and as a student of compulsory sickness insurance, these questions are in the nature of questions that might be asked a doctor from anybody who presents the subject of socialized medicine to a group of those who are interested in the socialization of medicine or in voluntary medical care insurance or any method of paying for medical care.

The first question starts off by refuting the oft-quoted figures that medical care in this country was inadequate because five million men were turned down for the draft. He points out, and gives figures for those who were blind and deaf; legless and armless; who were mentally deficient; with skeletal defects such as club foot, congenital dislocations, and so on. When the figures are boiled down they go to show that rather than five million having preventable diseases, who were turned down by the draft, the number actually amounts to slightly over a million and of this million there were innumerable men who were capable of being on a big league baseball team and engaging in other strenuous activities, who were classed as 4-F. The answers to the first two questions are indicative of the answers that are given throughout the book. They are based on legitimate statistics and are in part also the expression of opinion of a thoughtful individual. The last question; Mr. Walch points out that he has given deep thought to the subject because he is interested in his own health and that of his family. He feels that there are other ways to improve the medical care of the American people without having to resort to government control.

We are quite enthusiastic about this pamphlet, so much so that we are taking the liberty to suggest to the Executive Committee that at least one copy be sent to every doctor in the state.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

IMPORTANT ANNOUNCEMENT

The proposed plan for medical service for veterans in Louisiana which was submitted to the Veterans Administration by the executive officers of our State Society has been approved and became effective December 1. Full details and schedules of fees will be sent to each member of the Society by the Veterans Administration.

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PREPAYMENT PLAN RECEIVES SEAL OF ACCEPTANCE OF A.M.A.

Plan also approved for membership in Associated Medical Care Plans

The Louisiana Physicians Service Inc., received the Seal of Acceptance of the Council on Medical Service of the American Medical Association at a recent conference of the Council held in Chicago. The announcement was made by Frank Lais, Jr., Executive Director of the prepayment plan.

In addition to receiving the Seal of Acceptance the prepayment surgical and obstetrical care plan sponsored and approved by the Louisiana State Medical Society was accepted as a member of the Associated Medical Care Plans, Inc. This organization will handle the technical problems of the plan administration on a national basis and is an organization of the Plans themselves with headquarters in the Council on Medical Service's office in the A. M. A. Associated Medical Care Plans will work with the Council and will assist the Plans on such problems as reciprocity, sales training programs, standard policies for recording data and other administrative details.

Representatives from Louisiana, who attended conferences held in Chicago during the meetings of the Secretaries and House of Delegates recently, were Dr. Rhett McMahon, President of the State Society, Dr. P. T. Talbot, Secretary-Treasurer of the State Society and Mr. Frank Lais, Jr. Delegates to the A. M. A. House meetings were Dr. J. Q. Graves and Dr. M. D. Hargrove.

The Council on Medical Service of the A. M. A. reported to the House of Delegates the progress made by the voluntary prepayment medical care plans since the House meeting in Chicago in December, 1945. Seventeen plans in fifteen states were organized in 1946. This brings the total number of plans to over eighty spread out over thirty-three states, Hawaii and Puerto Rico. In addition thirteen states and the District of Columbia are in various stages of plan development, leaving only two states with no program in progress. The rapid growth in the number of plans will necessarily give impetus to the growth in enrollment. Already enrollment in many of the plans less than a year old exceeds 50,000 and several plans are approaching the 100,000 mark. In December, 1945, only two plans had over 250,000 enrollment; at present five of the plans are in this group, with one nearing the million mark and three approaching 500,000. Approximately 5,000,000 persons were enrolled in prepayment plans throughout the nation at the close of the year.

The council reported it will continue to concentrate on the over-all national picture as it pertains to voluntary prepayment medical care plans.

Regional conferences have been planned for prepayment plan executives and from all indications Louisiana may be selected as regional headquarters for meetings for the Gulf States area.

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DOCTORS—THEN AND NOW BROADCAST

On Saturday, January 18, 1947, 3:00—3:30 P. M. (CST) the American Medical Association in cooperation with the Council on Medical Service of the Louisiana State Medical Society presents, through the facilities of the National Broadcasting Company over WSMB, New Orleans, and other NBC stations in Louisiana, a broadcast dramatizing the life of Dr. Francois Marie Prevost. It is reported that Dr. Prevost was the first surgeon successfully to perform the Ceasarean operation in Louisiana, and as far as known, the second to accomplish this feat in the United States.

This program is part of a series of radio broadcasts tracing highlights in medical progress in the United States within the past one hundred years.

Doctors—Then and Now is a departure from the programs of recent years and offers dramatic stories from all sections of the country. It will bring to the microphone physicians from various regions who will tell up-to-the-minute news of what is being done to further medical knowledge and public health.

At the close of each dramatization the program will be switched to an NBC station in one of the designated sections. A physician who is an authority in that part of the country and who has been selected by his State Medical Society will present the picture of that area as it is today.

Speaking on the "Doctors—Now" portion of the program on January 18 will be Dr. King Rand of Alexandria to present the picture of medicine in Louisiana as it is today.

REPORT ON THE MEETING OF THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION, HELD IN CHICAGO, ILLINOIS, DECEMBER 9-11, 1946

The House of Delegates was called to order on Monday, December 9, 1946 at 10 a. m. with 167 members present. We were fortunate to have Dr. T. C. Routley, Secretary of the Canadian Medical Society to address the meeting on Monday. Dr. Routley has been a regular attendant at the meetings of the House of Delegates for a number of years. Recently he had attended the World's Health Conference in London, England. He brought a very inspiring message stressing the position that medicine or doctors must take in any future plans for world peace.

Admiral Boone reported to the House of Delegates on his recent survey of the hospital facilities and sanitary conditions in the coal mines. Apparently the different sections varied considerably in the extent and effectiveness of these measures. Generally, though, hospital facilities and sanitary conditions were not up to standard. In some places even elementary measures of control were not being enforced. Correction of these conditions obviously is the duty of all parties concerned, employees, employer and the medical profession.

There were a number of resolutions presented to the House of Delegatess. One of them was with regard to the political activities of the Surgeon General of the Public Health Department, Dr. Parran. The resolution, requesting that steps be taken to prohibit political activities on the part of the United States Public Health Services, was passed almost unanimously.

There was also introduced a resolution relative to the position of general practitioners in hospitals. It was pointed out that the present policy of permitting diplomates of the various specialty boards to dominate the staffs of hospitals was working an injustice on the other members of the profession. Of the 180,000 physicians in the United States there are approximately only 22,000 diplomates of the specialty boards. Recently the House of Delegates has established an individual section on the general practice of medicine which registered 939 members at the San Francisco meeting. The resolution as introduced and passed was to the effect that the House of Delegates of the American Medical Association suggests to the Council on Medical Education and Hospitals and directs it to further the realization of this suggestion, to wit: The requirements for approval of hospitals shall be so defined that there shall be (1) adequate protection of all doctors and their patients in obtaining hospitalization to the end that general practitioners as well as specialists shall have access to and use of hospital facilities; (2) that the criterion of whether a doctor may be a member of a staff or head of a department shall be his actual ability as a doctor and not dependent on special society or board membership; (3) that the American College of Surgeons be urged to conform to these general policies in their procedures in the standardization of hospitals; (4) that the American College of Physicians be urged to support the policy.

An important consideration of the House of Delegates at this meeting was a study of the survey of the A. M. A. made by the Raymond Rich Associates. They had been engaged to make a complete survey of the departments of the A. M. A., which was quite complete and in considerable detail. The various recommendations were adopted largely as recommended, although there were some changes made at the suggestion of the Board of Trustees and the House of Delegates. It is impossible to go into this report in detail. The essential thing in their report was that the A. M. A. must by means of better publicity and public relations sell themselves to the American people. They stressed the fact that most of the worthwhile work which has been done by the A. M. A. is unknown to the average American citizen, who apparently looks upon the A. M. A. simply as a protector of the economic rights of the doctor. The A. M. A. must convince the public that it is (1) seeking the truth as honestly in the economic and social aspects of medicine as it is in the scientific; (2) must put the people first, that is, must deal first and foremost with the needs of the public and second with the welfare of the physician; (3) the performance of the A. M. A. must be adequate. This can only be accomplished by better and more satisfactory public relations under the control of an individual specializing in this work. This phase of their recommendation is at present time being put into effect.

They also felt that the A. M. A. should not hesitate to support or oppose measures detrimental to the medical welfare of the country. That the A. M. A. should approach and work with all groups interested in health matters and not assume an isolated position.

The various publications of the Association were likewise studied and recommendations as to certain changes were suggested. They stressed the fact that Hygeia offered a splendid opportunity for better public relations. Through this journal, the circulation of which should be vastly increased, the public could be informed more satisfactorily about the A. M. A.

The recommendations made and those that are now being put into force will necessitate a considerable enlargement of the force at the home office and an increased expenditure for salaries and other expenses. It was generally felt that if by this increased expenditure the work of the A.M.A. could be improved, then this money would have been well spent.

Another important item of business was a report of the committee on the proposed revision of the By-Laws. This committee reported to the House of Delegates on December 10, but since there were so many suggestions made to the committee by members of the House the committee requested that they be given more time and be allowed to report at the June meeting in Atlantic City.

The Council on Medical Service reported on the progress of voluntary prepayment plans. It was pointed out that only two states have not started on a plan. It is expected that the enrollment will reach 5,000,000 early next year and it is hoped that with more plans in effect the enrollment should increase rapidly.

The importance of the Hill Burton Law should be impressed on all doctors. State and county medical societies must take an active part in activating the provisions of this act. In that connection, it would seem wise for every state medical society to make its own survey of hospital facilities in its state so as to determine the need, rather than have this done by some lay organization.

The general plan of the meeting in Atlantic City, the Centennial Celebration, was presented. Every effort is being put forth to make this the greatest meeting of the A. M. A. The program is being carefully prepared and is to include lectures, symposia, and panel discussions on timely topics so as to cover a wide field, thus appealing to every group. Representatives from various foreign countries are to be present. Certainly it promises to be an outstanding meeting, that we should make every effort to attend.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR OF MEETINGS

Jan. 13. Installation meeting, Orleans Parish Medical Society, Jung Hotel 7:30 p. m.
Jan. 15. Clinico-pathologic Conference, Marine Hospital, 7:30 p. m.
The Orleans Society of X-ray Technicians, 7:30 p. m.
Charity Hospital Surgical Staff, 8 p. m.
Jan. 20. Hotel Dieu Staff, 8 p. m.
Jan. 21. I. C. R. R. Hospital Staff, 12:30 p. m.
Charity Hospital Medical Staff, 8 p. m.
Jan. 22. Catholic Physicians' Guild, 8 p. m.
French Hospital Staff, 8 p. m.
Jan. 23. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.
Jan. 24. L. S. U. Faculty Club, 8 p. m.
Jan. 27. Scientific meeting, Orleans Parish Medical Society, 8 p. m.
Jan. 28. Baptist Hospital Staff, 8 p. m.
Jan. 29. Clinico-pathologic Conference, Marine Hospial, 7:30 p. m.
Jan. 31. New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
Feb. 3. Board of Directors, Orleans Parish Medical Society, 8 p. m.
Feb. 4. Orleans Parish Radiological Society, 7:30 p. m.
Eye, Ear, Nose and Throat Staff, 8 p. m.
Feb. 5. Mercy Hospital Staff, 8 p. m.
Feb. 6. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Executive Committee, Baptist Hospital Staff, 8 p. m.
Feb. 7. Ochsner Clinic Staff, 8:30 p. m.

N. J. Tessitore, M. D.,
Secretary.

EYE, EAR, NOSE AND THROAT BUILDING FUND COMMITTEE

Dr. Lucian W. Alexander has been named the head of the medical staff committee of the $300,000 building fund campaign for the Eye, Ear, Nose and Throat Hospital of New Orleans. The new addition to the present building will be constructed at a cost of $600,000 with $300,000 already available. When Mrs. Marie Celeste Stauffer Eastwick made her will in 1936, she bequeathed $1,200,000 as an everlasting trust fund to the hospital, the income to be used for maintenance and research, particularly in the field of the curable blind—and another $300,000 for a new wing for the overcrowded hospital.

It was 57 years ago that Dr. A. W. De Roaldes with the aid of a few public spirited citizens established the first Eye, Ear, Nose and Throat Hospital in rented quarters. It was in 1908 that the first unit of the present building was erected with funds given as memorials, such as the Hippolyte Laroussini Memorial Pharmacy. Then in 1922 the hospital had again outgrown its quarters and the second addition was added, this section known as the John Dibert Memorial. Now these two buildings are entirely inadequate to meet the present needs of the hospital.

The new addition will increase the bed capacity from 85 to 130. There will be a greater number of rooms for the various clinics. Five new operating rooms, one fenestration operating room, one bronchoscopic and biplane fluoroscopic operating room will have the latest scientific equipment. More teaching rooms and four new laboratories, including one for research work will be added. The teaching program offered in cooperation with Tulane University Graduate School of Medicine will be greatly facilitated with expanded quarters and equipment including a new medical library and two operating room observation galleries. The entire hospital will be air-conditioned.

Dr. Alexander pointed out, "The bequest of $1,500,000 will be lost by the Eye, Ear, Nose and Throat Hospital unless the $300,000 is raised now to construct the new hospital addition."

NEWS ITEMS

Dr. Emile A. Bertucci, Sr., spoke on "Music in Its Relation to Medicine" at a recent meeting of the Catholic Physicians' Guild at the Notre Dame Seminary. The talk was climaxed by the presentation of percussion instruments with sound movies.

Dr. Wm. B. Clark recently returned from Guatemala where he concluded the research project on onchocerciasis that he began last year.

At a recent meeting of the Catholic Daughters of America Dr. Walter J. Otis spoke on "Safeguarding Mental Health."

Dr. Daniel J. Murphy spoke on the "Evils of Government Controlled Medicine" at the annual state conference of the Louisiana Society for Crippled Children, in New Orleans, November 30, December 1.

INTERNATIONAL COLLEGE OF SURGEONS

The following members of this Society attended the eleventh assembly and convocation of the United States Chapter of the International College of Surgeons held in Detroit, October 21-23:

Dr. C. Bellone, C. J. Brown, Ernest Celli, Joseph Cohen, J. A. Colclough, Vincente D'Ingianni, C. M. Johnson, P. A. Phillips, Felix Planche, Wm. Roeling, Theo Simon, I. Tedesco, N. J. Tessitore, Jos. Vella, and Edwin L. Zander.

Dr. Zander presented a paper, written by himself and Dr. D'Ingianni, on "Case Reports and a Review of Literature on Endometriosis as a Cause of Small Bowel Obstruction."

RADIO PROGRAMS

The radio programs conducted by this Society and the Civic Affairs Committee of the New Orleans Association of Commerce came to a close with the November 25 presentation. The time given to us by radio station WDSU has been withdrawn because of other commitments.

We thank those of you who made these programs possible.

MEDICAL TOASTMASTERS

At a recent meeting of the Medical Toastmasters Dr. J. A. Colclough was elected president.

Other officers elected were as follows: Dr. Chas. L. Cox, vice-president; Dr. L. J. O'Neil, secretary-treasurer; and Dr. John B. Gooch, sergeant-at-arms.

SEPARATED FROM MILITARY SERVICE

Dr. Rufus H. Alldredge and Dr. Harry Morris were recently separated from military service.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

EIGHTH DISTRICT MEDICAL SOCIETY

The Eight District Medical Society met at the Bentley Hotel on December 12, for the purpose of electing officers for the coming year. Dr. Jack Cappel was elected President; Dr. Morris Hair, Lecompte, 1st Vice President; Dr. W. F. Couvillion, Marksville, 2nd Vice President; Dr. Herman C. Quantz, Secretary-Treasurer; Dr. J. Cappel, Delegate, Dr. R. E. Miller, Alternate.

The next meeting of the 8th District Medical Society was called for the first Monday in February at which time Dr. Masterson will present a paper on "The Rh Factor".

Herman C. Quantz, M. D.,
Secretary-Treasurer,
8th Dist. Med. Society.

THE LAFOURCHE VALLEY MEDICAL SOCIETY

A banner meeting of the Lafourche Valley Medical Society was held on December 11 in Raceland at the Rose Room of the O. S. T. Restaurant. The speakers for the evening were Dr. Edgar Hull who addressed the Society on "An Evaluation of the Electrocardiogram; Some Things to Expect and Not to Expect from the ECG." Following Dr. Hull's talk, Dr. Peter Graffagnino discussed "Office Gynecology." The annual dinner was a real feast which was apparently enjoyed by everyone.

Philip A. Robichaux, M. D.,
Secretary-Treasurer.

BI-PARISH MEDICAL SOCIETY

The Bi-Parish Medical Society met in the home of Dr. and Mrs. E. M. Toler. After an excellent dinner prepared and served by Mrs. Toler and friends the society was called to order in regular session.

Election of officers for 1947 resulted as follows:

Dr. Paul Jackson, President.

Dr. L. S. Nolan, Vice President.

Dr. E. M. Toler, Secretary-Treasury.

Dr. E. M. Toler, Delegate to the Louisiana State Medical Society.

Dr. E. M. Robards, Alternate.

Dr. C. S. Toler, Delegate to the 5th District Medical Society.

The society had as their guests, Rev. Harold D. Sadler, Vice Chairman of the Arkansas State Hospital, Drs. N. T. Hollis and A. C. Watson of the Arkansas State Hospital.

Dr. Rhett McMahon of Baton Rouge, Drs. Hollis, Watson and Alden were elected honorary members of the society.

Dr. Rhett McMahon of Baton Rouge, President of the Louisiana State Medical Society was the guest speaker of the evening. He delivered a very interesting discussion of the aims of the Louisiana State Medical Society during his tenure of office for the coming year.

A vote of thanks was extended to Dr. and Mrs. E. M. Toler for entertainment of the Bi-Parish Medical Society. The society adjourned to meet in the East Louisiana State Hospital the first Wednesday in March, 1947 at 7:30 p. m.

E. M. Toler, M. D.,
Secretary.

WEBSTER PARISH MEDICAL SOCIETY

The Webster Parish Medical Society met December 10, 1946 at the Minden Sanitarium. Supper was served and the following officers for 1947 were elected:

President: Dr. B. L. Cook.
Vice-Pres.: Dr. William R. Garrett.
Sec.-Treas.: Dr. E. S. Rogers.
Delegate: Dr. S. M. Richardson, Jr.
Alt. Delegate: Dr. T. A. Richardson.

TOURO INFIRMARY

The regular meeting of the Medical Staff of Touro Infirmary was held on Wednesday, December 11 at 8 p. m. The following program was presented: Clinico-pathologic conference by the Department of Pathology with a clinical discussion by Dr. Gilbert C. Anderson. Dr. Waldemar R. Metz presented two unusual cases of plastic surgery. Dr. Robert A. Katz gave a presentation on impending ischemic gangrene: A critique covering two years' trial with diethyl-ether, which was discussed by Dr. John DiLeo.

EXTRA RATIONS FOR PATIENTS

It has been brought to our attention that the forms for additional ration products previously sent to the "New Orleans District Medical Advisory Committee" should *now* be sent to the ration board in the patient's vicinity. By complying with the above request you will expedite the patient's receiving the additional rations and prevent the delay in mail transportation from the New Orleans District Medical Office back to the origin of the patient's request.

NATIONAL CONFERENCE ON MEDICAL SERVICE

The 20th Annual Meeting of the National Conference on Medical Service will be held at the Palmer House, Chicago, Illinois, on February 9. Registration will commence at 9:00 a. m. and the program will include discussions in the fields of national affairs, economics and medical education. All physicians are invited to attend; there is no registration fee. Dr. Cleon A. Nafe, Indianapolis, is President of the Conference and Creighton Barker, New Haven, is the Secretary.

LOUISIANA STATE UNIVERSITY SCHOOL OF MEDICINE NOTES

The following appointments to the faculty of the School of Medicine have not been previously announced in the journal. They include:

Dr. DeWitt Franklin Mullins, Jr., joined the faculty on October 3, 1946 as assistant professor of Pathology. Dr. Mullins is a graduate of Emory University, School of Medicine, and was recently released from active duty in the U. S. Army Medical Corps, where he was a member of the staff of the Army Institute of Pathology.

Dr. Florence L. Evans, who was associated with the School of Medicine, Louisiana State University, as instructor in Bacteriology, 1934-36, has been reappointed as assistant professor of Bacteriology. Dr. Evans received her Ph.D. degree at the University of Illinois in 1931 and during the past 10 years has been a member of the staff of the U. S. Public Health Service. During most of this period she has been engaged in research on leprosy.

Dr. George W. Hamlett was appointed assistant professor of Anatomy on September 1, 1946. Dr. Hamlett received his Ph.D. degree from the University of Texas in 1927 and has been associated with the University of Indiana, Harvard Medical School and the U. S. Biological Survey. His research has been in the field of embryology.

Dr. Daniel C. Darrow, professor of Pediatrics, School of Medicine, Yale University, lectured on "The Movement of Electrolytes in Relation to Body Water" at the School of Medicine on November 18.

Dr. Chapman Reynolds, associate professor of Pharmacology, gave a series of lectures at the medical schools of the University of Texas, Baylor University and the Southwestern Medical Foundation, under the auspices of the Phi Chi Endowment Corporation, during the period November 18 to 21.

SOUTHEASTERN SURGICAL CONGRESS

The Fifteenth Annual Assembly of the Southeastern Surgical Congress will be held in Louisville, Kentucky, Brown Hotel, March 10, 11, 12, 1947. The following physicians will appear on the program:

Claude S. Beck, Cleveland, Ohio; Wm. F. Rienhoff, Jr., Washington, D. C.; Gordon S. Fahrni, Winnipeg, Canada; Donald T. Imrie, Vicksburg, Miss.; Henry W. Cave, New York City; Leonard Edwards, Nashville, Tenn.; Hugh A. Gamble, Greenville, Miss.; K. M. Brinkhous, Iowa City, Iowa; J. O. Ranklin, Wheeling, W. Va.; R. B. McKnight, Charlotte, N. C.; Virgil S. Counseller, Rochester, Minn.; T. C. Davison, Atlanta, Ga.; M. A. Gilmore, Parkersburg, W. Va.; Sumner L. Koch, Chicago, Ill.; Marshall L. Michel, New Orleans, La.; Robert S. Dinsmore, Cleveland, Ohio; W. Perrin Nicolson, Jr., Atlanta, Ga.; Harry Morris, New Orleans, La.; Norman F. Miller, Ann Arbor, Mich.; Joseph Cunningham, Birmingham, Ala.; Duncan McEwan, Orlando, Fla.; Arnold S. Jackson, Madison, Wis.; John A. Martin, Montgomery, Ala.; Warren H. Cole, Chicago, Ill.; W. Milton Adams, Memphis, Tenn.; Irvin Abell, Louisville, Ky.; Elmer L. Henderson, Louisville, Ky.; A. J. Buist, Jr., Charleston, S. C.; Frank A. Hoshall, Charleston, S. C.; Joseph Stewart, Miami, Fla.; Everett I. Evans, Richmond, Va.; Thomas L. Lee, Kinston, N. C.

POSTGRADUATE COURSE—THE AMERICAN COLLEGE OF RADIOLOGY

The second annual Philadelphia postgraduate seminar in radiology will be held next spring, March 30 to April 4. The course is jointly sponsored by the Commission on Education of the American College of Radiology and the Philadelphia Roentgen Ray Society. Preliminary details have been announced by Dr. Barton R. Young, chairman of the special committee of the Philadelphia Roentgen Ray Society. Dr. Paul C. Swenson, Philadelphia, has been appointed director of lecture sessions. Dr. Calvin L. Stewart is secretary.

DIRECTORY OF CONVALESCENT HOMES

The Committee on Public Health Relations of The New York Academy of Medicine is preparing a new edition of the "Directory of Convalescent Homes in the United States," to be published by the Sturgis Fund of the Winifred Masterson Burke Relief Foundation of White Plains, New York. The last issue of this Directory was published by the Sturgis Fund in 1931 under the auspices of the Committee on Convalescent Care of the American Conference on Hospital Service and has long been out of print. The need of a new directory is recognized by all concerned.

Early in January, a questionnaire will be distributed to all recognized convalescent homes maintained by municipalities, counties or voluntary bodies. The information sought deals with the physical plant, the staff, the requirements for admission, the type of patients, therapy, and such other matters as will make the Directory a dependable guide for physicians who wish to refer their patients to institutions for convalescence. The questionnaire is so devised that it can be filled out with a minimum of effort. Convalescent homes that do not receive a questionnaire are urged to request one by writing to E. H. L. Corwin, Executive Secretary, Committee on Public Health Relations, The New York Academy of Medicine, 2 East 103rd Street, New York 29, New York.

PHARMACOPOEIA NOTES

The coming U.S.P. XIII will recognize only one iodine tincture, the two per cent, and has deleted the seven per cent solution. What was formerly called mild tincture of iodine will now be known as *iodine tincture.*

This action was taken because it is believed by physicians that the simpler preparation is not only equal to but superior to the older product as a first-aid dressing, which is the use for which iodine tincture is most frequently employed.

Pharmacists should call the attention of their customers to this U.S.P. change and explain the merits of the U.S.P. XIII iodine tincture. They should promote its use wherever possible, calling attention to the fact that the new product will not smart and sting as of old, and that in germicidal action it is equal to or superior to other first-aid preparations frequently used.

DOCTOR WANTED

The Town of Colfax, Louisiana (Grant Parish) needs the services of a general practitioner. For full particulars contact the Secretary of the State Society or the Lions Club, Colfax (Mr. C. Aswell Rhodes, Secretary-Treasurer).

A. M. A. CENTENNIAL

The American Medical Association is going to celebrate its centennial in Atlantic City, June 9-13, 1947. Elaborate plans are being made for this celebration.

Only Fellows and Invited Guests are eligible to attend. Membership in your state society is the primary qualification for Fellowship in the A. M. A. Fellowshiip dues and subscription to the J. A. M. A. are both included in one annual payment of $8.00, which is the cost of the Journal to subscribers who are not Fellows.

If you are not a Fellow and plan to attend the Atlantic City session, which will be a milestone in medical history, you can save yourself considerable time and confusion when registering, if you will write now to the American Medical Association, 535 North Dearborn Street, Chicago 10, and ask if you are eligible to become a Fellow.

SICKNESS INSURANCE

The Public Relations Bureau of the Medical Society of the State of New York has prepared a pamphlet entitled "Check and Double Check on Sickness Insurance." The subject is covered in 133 questions and answers with a complete index. This pamphlet is most comprehensive and any speaker or writer can find in a few minutes time material for a 15-minute talk. The pamphlet costs 25 cents with considerable reduction when a large number of copies are ordered. The pamphlet may be ordered from the Medical Society of the State of New York, 292 Madison Avenue, New York 17, New York.

INFECTIOUS DISEASES IN LOUISIANA

In the weekly morbidity report for the State of Louisiana for the week ending November 2, the following diseases in numbers greater than 10 were reported to the Louisiana State Department of Health: Cancer 122, unclassified pneumonia 100, tuberculosis 21, pneumococcic pneumonia 17, hookworm infestation 16 and poliomyelitis 10. The poliomyelitis cases were diffused over the state with Orleans Parish reporting three and East Baton Rouge two. For the week ending November 9, there were only three diseases reported in numbers greater than 10, namely hookworm infestation with 16, chicken pox 13, and diphtheria 12. Only four cases of poliomyelitis were reported this particular week. The drop in the cases of poliomyelitis, however, was only temporary as the succeeding week which closed November 16 showed 13 such cases reported from various parishes all over the state. Other diseases that occurred in numbers greater than 10 included pulmonary tuberculosis 39, typhus fever 18, and diphtheria 13. Practically all of the typhus fever cases were reported from Orleans Parish. The week which terminated November 23 showed the to be expected large number of cases of influenza. This week there were reported 130 cases which added to the previously reported cases for the year 1946, make influenza by far the commonest of reportable diseases, excluding venereal diseases. So far this year there have been reported over 20,000 such cases, measles coming next with slightly over 3,000 cases. Other diseases which occurred in numbers over 10 included 53 cases of pulmonary tuberculosis, 37 of septic sore throat, 29 of hookworm disease and 22 of malaria. Fifteen of these cases of malaria were known to have been contracted within Continental United States. Cold weather apparently had an effect on the incidence of poliomyelitis as only three cases were discovered in the state this week. For the week which closed November 30 there were reported a surprisingly large number of cases of cancer, 134, and of unclassified pneumonia, 112. The only disease that occurred in numbers greater than ten were influenza with 24 cases, pneumonia with 20, septic sore throat 14 and pulmonary tuberculosis 12. There were five cases of polioymelitis reported this week.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending October 31, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	29	527	556
Gonorrhea	1259	14503	15762
Granuloma inguinale	11	241	252
Lymphopathia venereum	6	91	97
Syphilis	1673	15340	17013

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce reported that for the week ending November 9 there were 137 deaths in the city of New Orleans, divided 90 white, 47 colored. Of these deaths, 24 were in children under one year of age, 14 white and ten colored. In the previous week there were reported only 118 deaths in the city and in the succeeding week which ended November 16 there were likewise comparatively few deaths, only 119 of the citizens of the city died, 73 of whom were white and 46 of whom were colored. The infant deaths numbered 14. The death rate increased the week ending November 23, there being 131 deaths in the city apportioned 94 white, 37 colored, with only 12 infant deaths. There was a further increase in the number of deaths that occurred the following week; eighteen more people died than the previous week. The 149 deaths were divided 106 white, 43 colored. Seventeen of the deaths were in children under one year of age and 14 of these were white children.

CORRESPONDENCE

December 3, 1946

Dr. P. T. Talbot, Secretary
Louisiana State Medical Society
1430 Tulane Ave.
City 13.

Dear Dr. Talbot:

On motion passed by the Second District Medical Society, we would like for you to know that we approve the Insurance Plan adopted by the Louisiana State Medical Society. This endorsement will be so reported in our minutes.

Sincerely,
Second District Medical Society,
Philip M. Tiller, Jr., M. D.
Secretary.

November 15, 1946

Louisiana State Medical Society
1430 Tulane Avenue
New Orleans, Louisiana

Attention: Dr. P. T. Talbot, Secretary

Gentlemen:

Pharmacists in the Prescription Departments of the United-Rexall Drug Company's Liggett, Owl, Sontag and Renfro Drug Stores are now under instructions to label all refillable barbiturate prescriptions as habit forming and also with the barbiturate content per unit.

We want every physician to know that the appearance of this information on any such refills of his prescriptions is not a policy initiated by our company. We do it because the Food and Drug Administration claims that the absence of such information would constitute a violation of the Federal Food, Drug and Cosmetic Act.

We foresee that such information on the label of a prescription may be disturbing to some patients and that the physician may blame our pharmacists for intruding in relations between the physician and the patient.

In order to temper the abruptness of such warnings, we are also placing the following statement on barbiturates, "Medicine of this type should be taken only for the purpose and for the length of time which your physician prescribed it."

We will appreciate it if you will acquaint your members with this fact through your publications or any other way possible. We think that this will help to accord the relations between physician and pharmacist which is one of the keystones of service to the American public.

Very truly yours,
Joseph A. Hailer,
Merchandising Manager—
Pharmaceuticals

WOMAN'S AUXILIARY

News from Auxiliaries has been very scarce lately, but surely not from lack of activity.

Several Auxiliaries have been very busy circulating publicity and making all the necessary preparations in order to have as large a crowd as possible to hear Mrs. Rex Fortenberry speak.

Mrs. Rex G. Fortenberry, attorney of Beaumont, Texas, and national lecturer, at the request of the Louisiana State Medical Society and under the sponsorship of the Auxiliaries to Louisiana State Medical Society, made a state wide tour, speaking in Monroe on November 18, in Alexandria November 19, Baton Rouge November 20, and in Hammond, November 21 at the Southeastern Louisiana College Music Auditorium, with a reception immediately following in the Social Room; in New Orleans, November 25, with a reception following in McAllister Hall of Tulane University; on Tuesday, December 10, in Lake Charles, (Little Theater), on Wednesday, December 11, at Lafayette, in Southwestern College.

Mrs. Fortenberry's subject was "Socialized Medicine—What it Would Mean to You." She became interested in the subject of the Murray-Wagner-Dingell bills "due to the fact that medical care is so vital to the people of the United States that any proposed changes in the character of the service should be carefully studied by those who will be affected," she said.

Mrs. Fortenberry practices law with her husband in Beaumont, Texas. She has a Bachelor of Law degree from the University of Texas and two Bachelor of Music degrees from Northwestern University of Chicago. She was admitted to practice in the state of Texas in 1938 and to practice in the Federal courts in 1940.

She is a member of the State Board of the Texas Federated Women's Clubs, which she serves as chairman of legislation, and is affiliated with the American Association of University Women, Altrusa, Business and Professional Women's Clubs, Pan American Round Table, Daughters of American Revolution, and Music Study Club. She serves also on the Board of Campfire Girls and the Beaumont Child and Family Counseling Bureau.

It is a great privilege and indeed a pleasure to hear such a distinguished person and one so well informed on such legislation now before Congress which if passed will force socialized medicine on the public. The Auxiliaries—privileged to have Mrs. Fortenberry have been fortunate indeed, as well as the public to whom she spoke.

The members of these Auxiliaries deserve much praise too, for the fine work they have done for they have given much of their time to work untiringly, to make their meeting a success.

It is only with this type of information given by an informed speaker, such as Mrs. Fortenberry, that the public can be informed properly as to the evils that would be experienced should socialized medicine become a certainty.

PARISH MEDICAL AUXILIARY MEETS IN PONCHATOULA WITH MRS. SCOTT

The regular monthly meeting of the Woman's Auxiliary to the Tangipahoa Parish Medical Society met Thursday of last week at the home of Mrs. Glenn T. Scott in Ponchatoula.

The members were honored by a visit from the state president, Mrs. Arthur Long, of Baton Rouge. Also present was Mrs. Roy C. Young of Covington, a past president of the state auxiliary.

After a short business session Mrs. Long spoke to the group on various phases of auxiliary projects, stressing the importance of doctors' wives doing all they can to become informed and help inform the public of the danger they will encounter if legislation is passed that will force socialized medicine on them.

Following Mrs. Long's talk the group enjoyed several musical selections on the accordion, rendered by Miss Zoe Lansing of S. L. C.

The guests were then invited into the dining room, where refreshments were served. The table was most attractive with an unusual centerpiece of miniature dining room furniture. The tiny table was set with a miniature turkey and all the trimmings, carrying out the Thanksgiving motif. This was arranged on an oval mirror and covered with a glass canopy. The centerpiece was surrounded by yellow chrysanthemums, and yellow tapers burned in crystal candelabra. Mrs. Collins Lipscomb poured tea.

This was one of the most enjoyable meetings of the year, and in attendance were Mesdames M. C. Wiginton, Arthur Long, Roy C. Young, C. D. Alessi, S. J. Cali, S. J. Dileo, A. J. Feder, Chas. Genovese, A. T. Montagnino, Glenn T. Scott, Jr., J. DeLoach Thames, Marshall M. Scarle, Collins P. Lipscomb, and Miss Zoe Lansing.

Mrs. M. C. Wiginton,
Chairman,
Committee on Publicity.

BOOK REVIEWS

Gastroenterology: By Henry L. Bockus, M. D. Philadelphia, W. B. Saunders Co. 1946. Pp. 1091. Price $12.00.

The reviewer had the opportunity thoroughly to familiarize himself with the first two volumes of Bockus' group and had looked forward eagerly to acquisition of the final volume. This third volume has lived up to the qualifications of its two earlier partners and completes the most authoritative treatise on Gastroenterology available. This remarkably thorough series completed during the difficult war years shows none of the deficiencies common to publications of this period. A careful choice of collaborators is apparent. For this third volume devoted to the liver, biliary tract and pancreas, and secondary gastrointestinal disorders there were fourteen contributors. Bockus, however, did not permit himself to become an editor, he competently fills in gaps between his fellow authors.

This work is so meticulous that for accurate evaluation it demands student-like reading rather than haphazard review. I, therefore, have been slow in giving my report that I might dedicate time to a pleasing task I assumed.

Tumen is responsible for classical sections on the anatomy and physiology of the liver, jaundice and hepatic disease. He is probably the outstanding contributor.

Berk shows excellent clinical ability in handling his portions on biliary tract disease.

I am certain many will feel more attention should have been given splenorenal shunt surgery in cirrhosis with ascites. Perhaps the author does not favor heroic surgery.

Dozzi's management, alone, and in collaboration with Bockus on the pancreatic issue is commendable.

In a period in which functional disorders of the gastrointestinal tract of neuropsychiatric origin are so prominently spoken of it is a little disappointing to find such a brief and inconclusive section as Yaskin's 29 page contribution. Yaskin is too much the neuropsychiatrist looking at gastrointestinal manifestations rather than a clinician striving toward successful therapeusis.

Miller, not an unduly enthusiastic allergist, handles his subject concisely and practically to give a fair relationship of gastrointestinal manifestations to allergic precipitants.

Stroud with Billet competently handles cardiologic aspects.

The review of this volume has been so enjoyable that one regrets not commenting on each section individually, certainly there is no deficiency encountered. However, the work is so detailed that one could write on at length without giving full credit where it rightfully belonged.

These three volumes create an excellent gastroenterology reference for specialist, student and practitioner. It is hoped the author may be able to keep the work revised annually.

Gordon McHardy, M. D.

The Diagnosis and Treatment of Pulmonary Tuberculosis: By Moses J. Stone, M. D. and Paul Dufault, M. D., F. A. C. P. Philadelphia, Lea & Febiger. 1946. Pp. 325. eng. Price, $3.50.

This is the third edition of a manual originally published by Hawes and Stone and deservedly lives up to the traditions of its predecessors. Because it is compact and brief, the material is presented in simple, almost dogmatic, fashion: for a manual, this is not objectionable. The twenty-six well written chapters, each with a small bibliography appended, provide an adequate survey of the current teachings on pulmonary tuberculosis. While obviously not intended for the specialist in phthisiology or for the research worker, it is suitable for the senior medical student or for the physician who wishes to review the field of tuberculosis without encountering the less "practicable" data. The tenor of all statements made is conservative; each recommendation for diagnosis or therapy is supported by abundant clinical and experimental evidence. The illustrations are helpful. It should be as well received as were the preceding editions.

SYDNEY JACOBS, M. D.

Clinical Laboratory Diagnosis: By Samuel A. Levinson, M. S., M. D., Ph. D., and Robert P. MacFate, Ch. E., M. S., Ph. D. Philadelphia, Lea and Febiger, 1946. Pp. 971. 192 engravings and 15 plates, 7 in color. Price, $10.00.

The third edition of this valuable work merits commendation. The authors say that they have endeavored to present data "in such a manner as to afford a rapid review of the pertinent points of previous studies with an attempt to correlate the didactic subjects with clinical diagnosis". Their efforts have been successful. To this reviewer the most appealing feature of the work is the correlation of pertinent facts gleaned from the basic medical sciences with clinical symptomatology in connection with the description of each clinical laboratory procedure; because this has been accomplished, careful page-by-page perusal of the volume may be considered to represent an acceptable, albeit a brief, "refresher course" in important aspects of clinical medicine.

In general, only the preferred technic is presented in describing each important laboratory test; expensive or complicated methods of testing are not described even though degrees of accuracy which they allow may be greater than those achieved with simpler technics. There are chapters which describe clinical laboratory methods in diseases of the mouth, the stomach, the duodenum and related organs, the intestine, the kidneys; other chapters deal with metabolism, chemical analysis of the blood, hematology, immunology and serology, cerebrospinal fluid, transudates and exudates, general bacteriology, sputum, skin tests and other biological examinations, milk and water analysis, histologic technic, legal medicine and toxicology. One excellent chapter describes laboratory methods in pediatric practice. Another, introduced in this edition, describes laboratory procedures in tropical diseases. An appendix tabulates valuable data. Ninety-seven tables allow succinct treatment of especially important subject. The format of the book is pleasing.

A number of specific criticisms may be listed: (1) Pyorrhea alveolaris, the authors write, "is thought to be caused, in part at least, by the *Endamoeba gingivalis*"; no definite proof exists as to the pathogenicity of this ameba. (2) The statement "practically no absorption takes place from the stomach" is refuted by daily experience with alcohols, drugs, poisons and sugars. (3) Since so many extra-gastric organic entitics produce symptomatic gastric disturbances, the validity of the statement "the clinical symptoms of a gastric disturbance together with euchlorhydria usually indicate a neurosis" is called into question. (4) On page 77 "pylorus" appears as "pyloris". (5) For "*Trichuris trichiura*", which appears several times, a more acceptable term would be "*Trichocephalus trichiurus*". (6) On page 129 "*Echinococcus granulosus*" is improperly spelled "*Echinococcus granulosis*". (7) The Weltmann reaction is described at undue length and rather remarkable claims are made for the help which it affords in differential diagnosis and prognosis. On the other hand, a most useful and widely employed technic of blood examination, the copper sulphate method for the determination of specific gravities of blood and plasma, blood proteins and hemoglobin levels, is not presented. (8) On page 494 there appear these sentences: "Anemia is a symptom, not a diagnosis. It is characterized by a decrease in the hemoglobin of the blood below the normal amount." It is realized that these statements express the views of one school of hematologists in America; the reviewer, however, prefers to align his views with another school which holds that anemia may be a disease, e.g., pernicious (Addisonian) anemia, and that changes in number and morphology of erythrocytes should be taken into account in defining anemia. (9) The plate on page 532 which depicts "filarial larvae in the blood" lacks sufficient clarity to warrant its inclusion in a work of this nature. (10) On page 625 "Coccidioides" is mispelled "Coccidiodes". (11) The propriety of classifying bartonellosis as one of the rickettsioses is indeed doubtful. (12) It is regretted that the authors have not presented data on toxoplasmosis. (13) As an acceptable name for the organism causing scrub typhus, *Rickettsia orientalis* is to be preferred to *Rickettsia tsutsugamushi*. (14) An unduly poor prognosis is inferred for cases of kala-azar, whereas with appropriate therapy it is usually good. (15) In the description of advanced cases of African sleeping sickness there appears the erroneous statement: "Lumbar puncture is negative." (16) The most important arthropod vector of Chagas' disease is listed as "*Triatoma megista*"; an accurate designation is "*Panstrongylus megistus*." (17) In the discussion of malaria no mention is made of the exo-erythrocytic stage of malaria plasmodia in man. (18) On page 790 loose structure of a sentence allows the reader to understand that a microgamete of a malaria parasite consists only of a "flagellum" whereas, like a spermatozöon, it consists of a flagellum and a nucleated body. (19) Description of technics for staining thick blood films is not clear; the technics chosen for presentation are not modern. (20) In a discussion of granuloma inguinale there appears the remarkable statement: "clinical symptoms are absent." (21) In the material upon the rickettsial diseases the Neill-Mooser reaction, which appears in laboratory animals following inoculation of material from cases of endemic typhus fever, is not described; moreover, the valuable complement-fixation tests of the blood to differentiate the rickettsioses specifically are not presented. (22) The epochal studies of Sabin, Philip and Paul upon the leukocytes in sand-fly fever are not mentioned. (23) It is felt hat the present state of our knowledge does not warrant the unequivocal statement on page 817: "Trench fever is caused by the invasion of *Rickettsia wolhynica* into man." (24) On page 817 Oroya fever is misspelled "Oroyo" fever.

While the foregoing list of critical comments is lengthy, it is felt that the general excellence of the work is only slightly impaired by the need for their enumeration. Perhaps the reviewer may best describe this book as one which he will certainly acquire in order to have the benefit of consultation with its authors in his daily work.

In final comment on this volume special attention should be directed to the excellence of presentation of material upon the clinical laboratory study of specimens taken at biopsy from the bone marrow.

EDWIN S. KAGY, M. D.

Diseases of the Retina: By Herman Elwyn, M. D. Philadelphia, The Blakiston Co., 1946. Illus. Pp. 587. Price, $10.00.

Ophthalmology is indebted to the author for a difficult and important work well done. His presentation of the subject is less complete but simpler than that of Leber, in his two volume classic of thirty years ago. The brief case reports used to illustrate especially rare conditions, render a distinct service in making this volume easy to read. The excellent illustrations and photographs also facilitate understanding of the text. He has correctly emphasized that many degenerations are an exaggerated form of presenility, and that these follow definite patterns, in which ocular participation is often a part of a similar involvement in other parts of the body.

The classification followed materially clarifies the subject, although conflicts exist. Conditions such as essential arterial hypertension are discussed under circulatory disturbances, though they are basically constitutional degenerative diseases which are discussed in another section.

The number of summaries could have apparently been increased to advantage. A more detailed description of pathological sections would have been advisable.

The table of contents, bibliography, and index, are especially complete for a volume of less than 600 pages.

The author is to be congratulated on having made a major contribution to ophthalmology.

CHARLES A. BAHN, M. D.

Diseases of the Adrenals: By Louis J. Soffer, M. D. Philadelphia, Lea & Febiger, 1946. Pp. 304. Price, $5.50.

The author has presented in less than three hundred pages the accrued information in the anatomy, physiology and pathology of the adrenals, in addition to the clinical manifestations and management of adrenal disease.

The description of clinical syndromes is developed after an elucidation of the part these organs play in endocrine inter-relationships, electrolyte and water balance, carbohydrate and protein metabolism, the physiology of the circulation and shock.

Considerable space is given to Addison's disease, the Waterhouse-Friderichsen syndrome, the adrenogenital syndrome and neoplastic adrenal diseases. The term adrenogenital syndrome is used to include a variety of conditions which produce similar clinical states. It includes pituitary basophilism, adrenal cortical tumors and hyperplastic tumors of the anterior pituitary, arrhenoblastoma, granulosa cell tumor, thymus tumors, pineal tumors, and hyperostosis frontalis interna.

Neoplastic diseases of the adrenals and the chromofin system are discussed from a standpoint of the precurser cells of these tumors.

This book is intended for the clinician. It contains practical points in the recognition and management of adrenal diseases. Case histories, photomicrographs, and reproductions of microscopic sections illustrate the text. A section describing the technic of electrolyte determinations in serum and urine, male sex hormone and 17 ketosteroid excretion, the salt tolerance and deprivation tests, the water tolerance test and perirenal insufflation is a valuable source of reference. In addition to bringing together the present knowledge of the adrenals, the author has given frequent summaries and the benefit of his own experience in many instances. This book's patho-physiological approach to disease makes it interesting reading in a fascinating subject in medicine. The monograph meets a definite need and will undoubtedly be well received by the profession.

STANLEY COHEN, M. D.

Renal Diseases: By E. T. Bell, M. D., Philadelphia, Lea & Febiger, 1946, Pp. 1946. Price $7.00.

Dr. Bell has attempted to bridge the gap between the clinical manifestations of renal disease and their pathology by a study begun on the wards and followed to the microscopic study of kidney tissue sections. This information, collected over a period of 25 years is presented following a short resumé of normal renal anatomy and physiology.

Of particular interest are the studies in glomerular nephritis. Hemorrhagic nephritis is considered as a separate entity. These patients present hematuria but lack the proliferative endothelial and epithelial changes found in the proliferative type of active glomerular nephritis. When uremia develops in these patients Bell attributes it to extensive filling of the tubules with blood. The azotemia of the acute proliferative types of glomerular nephritis is ascribed to the obstruction due to endothelial proliferation of the glomerular tuft, and epithelial proliferation of the capsule (referred to as crescents). The term chronic azotemic glomerular nephritis is used to denote that phase of the disease in which hyalinization of the glomeruli, or fibrosis of the epitheliol crescents occur.

The pathogenesis of the so-called intercapillary lesion is described as due to hyalinization of the inner layers of the basement-membrane of the capillary wall. Fusion of fragments of the capillary wall ultimately lead to what appears to be an intercapillary hyaline mass. This interpretation is contrary to the opinion of other authors.

In the study of the nephroses, excellent photomicrographs demonstrate the difference between terminal basement membrane thickening of the "pure" type and the glomerular proliferation of the "mixed" type. Azotemia is shown to be related to either of these processes.

The renal lesions in diabetes, pyelonephritis, tubrenal arteriolsclerosis is presented in detail, with the conclusion that they are independent diseases that intensify each other.

The renal lesions in diabetes, pylonephritis, tubular disease, specific infections, hyperparathyroidism and gout are illustrated. Nephrolithiasis and tumors are discussed in detail.

This book is well written and profusely illustrated with photomicrographs to demonstrate points in the text.

Stanley Cohen, M. D.

Medical Education and the Changing Order: By Raymond B. Allen, M.D., Ph. D. New York, Commonwealth Fund, 1946. Pp. 142. Price $1.50.

This monograph is as delightful to read as it is informative. Naturally Dr. Allen made no attempt to exhaust an inexhaustible subject but it is remarkable how much that is stimulating has been put into so few pages. These pages contain not only a fine philosophical analysis of the relation of medical education and medical practice to communities in general but they also contain concrete suggestions for the closer integration of medical education with general education or, perhaps, vice versa. It is, of course, not desirable that every medical school should teach in the same manner but perhaps if every medical educator in this great country could read this charming treatise, there would, at least, be a common starting point for the efforts at supplying a curriculum. The author's wide experience in medical teaching and medical school administration is evident in every chapter and each page has been touched by the delicate hand of a literary artist.

H. W. Kostmayer, M. D

Peripheral Vascular Diseases: By Edgar V. Allen B.S., M.A., M.D., M.S., F.A.C.P., Nelson W. Barker, B.A., M.D., M.S., F.A.C.P., and Edgar A. Hines, Jr., M.D., B.S., M.A., M.S., F.A.C.P. Philadelphia: W. B. Saunders Co., 1946, Pp. 871. Price, $10.00.

This book is dedicated to Dr. George E. Brown and presents the observations made as well as the methods of treatment developed and advocated by the authors and Dr. Brown in the field of peripheral vascular diseases. In addition to the material presented by the principal authors the book contains subject matter prepared by eleven other contributors. From the standpoint of physiology, pathologic anatomy and diagnosis the volume is encyclopedic. The format is very good and there are many excellent illustrations as well as an abundance of pertinent references. In general, medical or conservative methods of therapy are emphasized. The surgical approaches and technics which have been found useful in the management of peripheral vascular diseases are also presented. Altogether, this book very well summarizes present day knowledge and practice in peripheral vascular diseases and is a real contribution to the field, valuable to both students and practioners.

Ambrose H. Storck, M. D.

PUBLICATIONS RECEIVED

The Commonwealth Fund, New York: Health Insurance in the United States, by Nathan Sinia, Dr. P. H., Odin W. Anderson and Melvin L. Dollar.

Lea & Febiger, Publisher, Philadelphia: Clinical Hematology by Maxwell M. Wintrobe, M.D., Ph. D.

W. B. Saunders Company, Publisher, Philadelphia: Muscle Testing by Lucille Daniels, M.A., Marian Williams, M.A., and Catherine Worthingham, M.A.

Charles C. Thomas, Publisher: Urgent Surgery, by Julius L. Spivack, M.D., LL. D. Gastroenterology in General Practice by Louis Pelner, M.D.

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SADDLE BLOCK ANESTHESIA*

ITS APPLICATION TO OBSTETRICS

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and
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NEW ORLEANS

Saddle block is a term used to designate a form of low spinal anesthesia confined to the perineal area. It differs from low spinal anesthesia induced in the ordinary methods in having a greater degree of controllability. Exact localization of anesthesia in the sacral segments with comparatively small doses of local anesthetic drugs may be obtained. The authors have employed the technic for rectal, urologic and gynecologic surgery for some time. The same extent of anesthesia obtained with caudal block is obtained with saddle block in a simpler, safer and more precise manner. By using longer lasting drugs such as pontocaine or nupercaine anesthesia and analgesia lasting two and three hours, respectively, may be obtained by a single injection. The recent popularization of caudal anesthesia for obstetrics by the continuous technic suggested to the authors that saddle block could be used instead and might be more desirable.

On the whole, spinal anesthesia has not been popular in obstetrics. One of the drawbacks has been that procaine has been considered the drug of choice. Although procaine is excellent for spinal anesthesia, its duration of action usually averages an hour. The injection must be repeated frequently; otherwise the drug can only be used for anesthesia for the delivery. The longer lasting drugs allow its use for analgesia during labor as well as for the manipulations of delivery. In view of the excellent results obtained with saddle block in surgery, there did not appear to be any reason why its use could not be extended to obstetrics. This is a report of our experiences in extending the use of the block in obstetrics. Nupercaine and pontocaine have been employed to provide the desired longer duration.

TECHNIC

The materials for performing a saddle block are the same as those used in spinal anesthesia. A wheal needle, a 20 gauge short bevel spinal needle and a 2 c.c. syringe are the necessary items. Glucose (10 per cent) in physiologic saline or distilled water, a 1/200 solution of nupercaine and 1 per cent procaine for infiltration are the necessary solutions and drugs. If pontocaine is desired the 1 per cent solution is employed.

Nupercaine, 0.5 c.c. of 1/200 solution is thoroughly mixed with 0.5 c.c. of 10 per cent glucose solution and drawn into one of the 2 c.c. syringes. The solution thus prepared contains 2.5 mgm. of nupercaine. Five milligrams of pontocaine must be drawn and mixed with the glucose if this drug is employed. No spinal fluid is necessary in the preparation of the fluid in either case. The patient is placed in the sitting position in the center of the operating or delivery table, or at the edge of the bed with

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legs dangling over the side. The puncture is simplified if the patient leans forward with her arms folded across the chest and her shoulders are supported by an assistant. An intradermal wheal is raised at the fourth lumbar interspace and the interspinuous tissues are infiltrated with 1 per cent procaine. The third interspace may be employed if the fourth is not accessible. There is little difference in the level of anesthesia obtained in either case. The syringe containing the solution is attached to the needle and aspiration is attempted to insure a free flow of spinal fluid. Not more than 0.1 c.c. of spinal fluid should be withdrawn into the syringe, otherwise dilution of the solution which causes spreading of the drug beyond the desired spinal segments occurs. The needle should be readjusted until a satisfactory flow of spinal fluid is obtained if it does not flow freely. Failures in spinal anesthesia can usually be ascribed to unsatisfactory lumbar puncture. It is advisable to prepare the solution in advance of the lumbar puncture to avert failures due to dislodgment of the needle by sudden unexpected movements of the patient. The solution is injected as rapidly as gentle pressure upon the plunger of the syringe permits—usually less than two seconds. The patient is then allowed to remain in the upright sitting position for thirty seconds after completion of the injection after which time she is placed in the recumbent position. The head is elevated on a pillow in order that the neck may be flexed. The solution should not be introduced during a uterine contraction because the spinal fluid pressure and currents established in the subarachnoid space force the drug into the thoracic region. The table may be flat or placed in a reversed Trendelenburg position at an angle of approximately 5°. The glucose causes the solution to be hyperbaric or heavier than spinal fluid. The drug is therefore carried downward to the tip of the dural sac. The anesthesia in these segments will be intense and well localized. It is well known that sensory and autonomic fibers of a mixed nerve are more sensitive to the effects of a given concentration of a local anesthetic drug than the motor. If the patient is promptly returned to the recumbent position a given dose of drug becomes distributed over more segments of the cord than if she remains sitting for some time. In either case, the sacral segments are exposed to a more concentrated solution of drug than the lumbar and both the motor and sensory fibers of these segments are affected. The perineal area is more completely anesthetized and relaxed. When the patient sits up sixty or more seconds there is no sensory anesthesia or motor paralysis in the legs and thighs. If the patient sits up 30 seconds, the lumbar segments are exposed to a weak concentration of solution of the drug. Sensory anesthesia of the legs and thighs with little or no paralysis of the leg and thigh muscle is obtained. In most cases the analgesia over the thigh gradually merges into an area of hypalgesia over the lower abdomen. This distribution is desirable for obstetrics because it also affects the sensory fibers of the uterus and cervix. The sensory fibers to the cervix and lower uterine segments are derived from the sacral and lower lumbar spinal nerves; those of the upper part and the fundus from the upper lumbar and twelfth thoracic segments. The motor innervation is still a matter of question. It is believed that the motor fibers are derived from the thoracic segments. Consequently, there is no diminution in the number or force of the contractions because the drug does not extend to this level. Pain from contractions disappears, but labor continues.

METHOD OF STUDY

The technic in this study was limited exclusively to normal primipara or multipara. Patients presenting any systemic abnormalities or obstetric complications were excluded. The block was performed after the inception of the first stage of labor—usually when the contractions appeared at three or four minute intervals and the cervix was 5-6 cm. dilated or approximately 80 per cent effaced. Sedatives were purposely omitted in order to evaluate the effects of the block alone properly. Blood pressure and pulse rate were recorded at three or four minute intervals, shortly after induction of analgesia, and at 10 to 15 minute intervals after the first half hour. The anesthetist remained in constant attendance closely observing the frequency and intensity of contractions, fetal heart tones, progress of labor, respiration, and other reactions of the patient. Apparatus for the administration of oxygen or artificial respiration were held in readiness as is the usual procedure in all cases of spinal anesthesia. If repetition of the block was necessary, the injection was repeated in exactly the same manner as the previous injection as soon as analgesia began to disapppear.

RESULTS

Satisfactory analgesia during labor and anesthesia at the time of delivery were obtained in the majority of cases. Nupercaine was used in 88 per cent of the cases. Pontocaine was chosen when less than two hours' analgesia seemed indicated. Anesthesia became established within two to five minutes and lasted an average of three hours with both drugs. The onset of anesthesia, when nupercaine or pontocaine are mixed with glucose, may require four or five minutes. In some cases using nupercaine, analgesia lasted as long as five hours. The average, however, was three hours with nupercaine and two hours with pontocaine. Seventy-seven per cent of the subjects experienced complete pain relief. Fifteen per cent complained of pain in the upper abdomen during the period of traction of the forceps. The pain was described as a dull ache in the epigastrium or along the back in the lumbar region. Inhalation of analgesic concentrations of nitrous oxide was sufficient to relieve this discomfort. Pain in the perineum during repair of the episiotomy was absent. Seven per cent complained of pain in the perineum or back or were not relieved of the pain from the uterine contractions. Failure to obtain satisfactory analgesia was ascribed to errors in technic. It is imperative that timing and measurements of volumes of solutions be precise if a successful block is to be obtained. The block was repeated after 15 or 20 minutes. Satisfactory analgesia was obtained the second time in all these. There is no objection to or danger in immediately reattempting the block in the event of failure because the doses employed are small.

The patients were comfortable and cooperative throughout the labor and delivery. Patients in pain or who were not cooperative before the block was performed, immediately became so. Patients were able to bear down when so instructed and aid in the delivery since the abdominal muscles were not paralyzed. The absence of paralysis in the legs and thighs allowed the patient to move about in bed and shift to desired positions. In some patients a mild paresis of the thigh and leg muscles was present but it did not interfere with motion, although it did limit it to certain extent. The analgesia in the legs and thighs was a desired feature because it alleviated discomfort when the lithotomy position was instituted during the delivery. Low forceps were used in 68 per cent of the patients. A single injection was sufficient in 80 per cent of all cases. In 30 per cent, labor outlasted analgesia and second or third injections were required. The shortest time from the institution of analgesia until delivery was 15 minutes; the longest 11 hours, 51 minutes. The injection was repeated twice in the latter case. The third stage of labor was not remarkably different from cases delivered with other types of analgesia or anesthesia. Postpartum hemorrhage was not encountered; excessive bleeding was no problem. The babies cried spontaneously and required no rescuscitation.

Nausea was present in 10 per cent of the patients; vomiting in 8 per cent. Nausea was most prevalent among patients who were given food or fluids during labor. This occurred early in the series and was a source of concern until the cause was recognized. It is advisable to restrict fluids and food during labor to avoid this complication. A reduction in blood pressure averaging 10 mm. mercury systolic was noted in 50 per cent of the cases. The lowering was attributed to removal of psychic effects or to the pain relief. A momentary systolic drop, sometimes as low as 80 mm.

mercury occurred shortly after completion of the injection. Recovery was, however, spontaneous and required no therapy other than deep breathing.

Hypotension with bradycardia is characteristic of spinal anesthesia, particularly when the block is extensive. However, it is unusual during saddle block. In this series it was absent in all but five cases. In these, the blood pressure fell below 80 systolic and continued at this level for some time. Ephedrine 15 mg. intravenously, restored the blood pressure to the preanesthetic level. The number of spinal segments affected in saddle block is few. Motor anesthesia is confined to a few small muscles. Physiologic changes, therefore, are minimal and compare in no way with those encountered in the usual type of spinal anesthesia in which numerous spinal segments are anesthetized. Respiratory embarrassment was not encountered in any case. Although hysterographic and other studies of the effects upon labor were not attempted, it was obvious at the outset that labor was retarded if the block was performed before strong regular contractions were present and the cervix was dilated. Likewise the impression was gained that the incidence of delivery by forceps was increased. If the block extends too high (analgesia of the thoracic nerves) labor is retarded.

Post spinal headache, backache, urinary retention, distention, ileus, meningismus, palsies or other complications were not encountered in the series.

DISCUSSION

Obviously, 156 cases is not a sufficient number to prove the merits or demerits of this or any other method of analgesia or anesthesia. However, the applicability of saddle spinal block to obstetrics is established. One must bear in mind that the usual objections, hazards, and complications of spinal anesthesia in a series as small as this is of no significance. It must be emphasized that these were normal cases, free from all complications and that the applicability of this method of analgesia to other than uncomplicated obstetrical cases requires further study.

Saddle spinal offers certain advantages over the continuous caudal block. The uncertainty of locating the caudal canal, the presence of an indwelling needle on catheter, the hazards of improper placement of the needle, the repeated use of relatively large volumes of local anesthetic solutions, the uncertainty of distribution of anesthesia are eliminated in this technic.

SUMMARY

Saddle block anesthesia is a form of low spinal anesthesia confined to the perineal area. A technic is described for prolonged analgesia using the longer lasting drugs—nupercaine and pontocaine. The block was used in 156 normal deliveries with satisfactory results. The applicability of this method of anesthesia and analgesia to obstetrics is demonstrated by its success in this series. The method is recommended when spinal anesthesia is desired or indicated in obstetrics. It possesses the advantage of certainty and simplicity over the continuous caudal technic.

THE PREVENTION OF Rh ISOIMMUNIZATION DUE TO BLOOD TRANSFUSION

J. W. DAVENPORT, JR., M. D.* †
New Orleans

In no phase of Rh investigation has more rapid advance taken place than in the development of procedures for detecting the various Rh antibodies, which are, of course, the actual serologic proofs of isoimmunization. Thanks to the efforts of a small number of investigators, and their associates, serologic laboratories today have at their command a number of excellent tests for the detection and identification of Rh antibodies. I have found these procedures to be most useful when employed in parallel. They include the agglutination and absorption tests of Levine[1, 2], the blocking and so-called conglutination tests of Wiener[3, 4],

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Diamond's slide agglutination procedure[5] and various modifications of each.

While the tools available for demonstrating Rh isoimmunization have thus increased both in numbers and accuracy, we are still largely without power to permanently inhibit or neutralize the antibodies once they have been proliferated. As is well known, the Rh complex is highly antigenic in some humans and Rh—individuals may become sensitized or isoimmunized following the entrance of Rh+blood into their circulations, either by transfusion, pregnancy, or both.

However, it is unusual indeed for an Rh—person to become sensitized following a single exposure to Rh+blood. Only about 15 per cent of the white race are Rh—and of this minority group not more than 5 per cent seem capable of being sensitized even following several exposures to the antigen (Rh+blood).[6] Most unfortunately, there is no method known for indicating which of the Rh—individuals will become sensitized and form antibodies if exposed to Rh+ blood. When one considers again that there is still no way yet known for safely deimmunizing sensitized persons, the vital importance of preventing isoimmunization becomes apparent. As far as pregnancy is concerned, it has been suggested that Rh typing should become a part of premartial blood tests and that Rh+men should be forbidden to marry Rh—women. This idea is ridiculous in light of the statistics on sensitization already mentioned and certainly unthoughtful of basic human rights.

Now, when we examine the sensitization problem from the transfusion approach, we are in better position to accomplish a useful end. The repeated transfusion of Rh+blood into Rh— recipients very infrequently produces isoimmunization. However, it does occur at times in both sexes although producing a less intense degree of antibody formation than Rh+pregnancies in Rh—mothers. The relationship between maternal Rh antibodies and the syndrome "hemolytic disease of the newborn" is well established and requires but passing mention at this time.*

While this syndrome, in one stage or another, is seen only in about one in 250 deliveries[7] more than 90 per cent of the time it is the result of the now classical relationship of Rh—mother and Rh+father and baby.[8] In these cases the maternal antibodies attack the fetal erythrocytes and set in motion a chain of patho-physiological events leading to the development of the hemolytic syndrome. Succeeding Rh+pregnancies are usually affected more severely.

Practically the only weapon we have at present for preventing Rh isoimmunization is the routine Rh typing of all patients who are to receive transfusions, ensuring that Rh—patients will receive Rh—blood. It is most essential that this routine test be done in the case of female patients, first because of the two routes of sensitization open via transfusion and pregnancy and secondly because in the case of women of childbearing age or younger, there are future offspring to consider. Indeed, from the viewpoint of possible sensitization a blood transfusion in a woman should be regarded as equivalent to a pregnancy. The routine typing must also include little girls and female infants. As will be shown later, the transfusion of Rh+blood into an Rh—girl can produce a sensitization which will persist for years and which will flare up on subsequent exposures to the antigen, leading to hemolytic transfusion reactions or to hemolytic disease of the newborn.

At this point it is in order to refer to a recent report published by Levine and Waller[9] concerning the incidence of erythroblastosis fetalis in first born children. This extensive study showed a higher degree of incidence and severity of erythroblastosis in the Rh+first born children of Rh—mothers where the mothers had a history of having received one or more blood trans-

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(*) The term "hemolytic disease of the newborn" is used to include erythroblastosis fetalis, icterus gravis neonatorum and hydrops fetalis, all three of which clinical entities stem from the same basic cause—extensive blood destruction in the fetus with excessive compensatory erythropoiesis.

fusions from a few months to several years before the first pregnancy. Significantly, in a group of Rh—mothers of affected Rh+ first born infants, although the mothers had negative transfusion histories, it was not possible to rule out completely the intramuscular injection of blood in these mothers during their own infancy. In this connection it must be stressed that the intramuscular injection of blood will sensitize a susceptible Rh—person practically as well as the intravenous injection.

In the case of older women the Rh type obviously should be determined, since this group may have been sensitized years before by transfusions or pregnancy and may suffer severe or even fatal reactions to otherwise compatible Rh+blood.

Certainly the ideal situation must and will some day be the routine Rh typing of all patients who are to receive transfusions, with Rh—blood only being given to the Rh—patients. Modern blood banks are struggling toward this goal but are hampered at present by the frequent difficulty of obtaining adequate supplies of reliable Rh testing sera and by the problem of maintaining an abundant supply of Rh—donor blood. However, as more and more Rh cases are studied, the serum situation is improving, and since most banks Rh type all their donors the number of known Rh—people in almost every community will increase.

Even in the most efficiently operated blood banks the demand for Rh—blood at times exceeds the supply. Since we feel that it is more essential that Rh—females receive Rh—blood, we do not routinely Rh type male patients. The procedure is done on men only when indicated by a history of repeated transfusions or of one or more unfavorable reactions to transfusion. In such cases the patient's serum is tested for Rh antibodies and if these are present, he is given Rh—blood. In my experience this policy has proven to be satisfactory in operation because it frees quantities of Rh—blood for the more urgent need of female patients. Further, in the space of the past three and one-half years, during the course of several thousand Rh examinations and blood transfusions, I have encountered but three cases in which the repeated administration of Rh+blood to Rh—males induced the formation of antibodies. In each case the antibodies were detected before any severe reaction occurred and subsequent transfusions were with Rh—blood.

As has been stated, once Rh isoimmunization has been established, there is nothing at present to be done to correct the situation. This presents no great obstacle to transfusion therapy other than obtaining suitable Rh—bloods for Rh—patients. Since Rh follows a definite genetic pattern within any family, we have at times been successful in obtaining additional supplies of Rh—blood by Rh typing close blood relatives of the patient.

Even though only a small percentage of Rh—people ever become sensitized, it is quite apparent that every effort must be made to prevent accidental sensitizations which might result from the transfusion of Rh+blood to Rh—recipients. Only by accurately Rh typing all patients who are to receive blood, can we hope to avoid instances of inadvertent isoimmunization with its sequelae of hemolytic reactions and/or complications of pregnancy. When one analyzes the Rh problem in relation to pregnancy, many far reaching ramifications may be perceived, for here we are dealing not only with the patient herself but must weigh the potential effect of maternal antibodies which may be induced by future pregnancies.

In selecting cases to include in this report, I have deliberately chosen those which can be held up as "horrible examples" of what may happen if a young female patient is given blood without regard to her Rh type. It must be said, however, that in two of the cases, the original transfusions were given some years before the discovery of the Rh Factor, while in one case, knowledge of Rh was very much in its infancy and no typing sera were available, even if we had realized that we should have Rh typed the patient.

CASE NO. 1

In 1936, the patient, a girl 12 years old, was admitted to another hospital with a diagnosis of streptococcic septicemia. While there she received a 500 c.c. indirect blood transfusion from her uncle, with no untoward reaction. No other blood was given and recovery was uneventful.

In August, 1944, now 21 years old and married, the patient was delivered of a full term male infant exhibiting marked jaundice and erythroblastotic anemia. Rh types and blood groups as determined by the writer were as follows: patient Rh — Group A; husband Rh+ Group A; infant Rh + Group A. In spite of a transfusion of 75 c.c. of compatible Rh — blood, the baby expired in about 48 hours. The original diagnosis of hemolytic disease of the newborn was confirmed by postmortem findings, most of the liver and a large portion of the spleen being replaced with the characteristic, accessory erythropoietic tissue.

The patient's serum was tested for anti-Rh agglutininins by the saline-cell suspension method. Results were negative. Undoubtedly blocking antibody would have been found at this time, had that test been known.

In February, 1945, the patient was referred back to the writer and in reviewing her history, the fact of the transfusion more than eight years previously was elicited. We were able to obtain a blood sample from the relative who had been the donor and found him to be Group A Rh +. The patient's serum was still negative for anti-Rh agglutinins but rather strong blocking antibody was found, in a titer of 1:8. The writer's serologic findings in this case were confirmed by Dr. Philip Levine who also very kindly determined the Rh genotype of the husband, establishing it as homozygous (Rh-Rh). This latter point made the prognosis for future pregnancies very poor. Since the couple wished to attempt another pregnancy, however, they were advised to wait for at least two years, not with the idea of causing a loss of sensitivity in the wife, but rather to permit the actual antibodies to disappear from her circulation.

Unfortunately, the patient became pregnant again within a short time. She was admitted to the hospital on October 2, 1945, and two days later was delivered of a six and a half month, macerated fetus which had been dead about six weeks. The patient had not felt life since the second week of the fifth month. Because the fetus was in a considerable state of decomposition, its blood being hemolyzed, the Rh type could not be determined. Undoubtedly it was actually Rh + since examination of the mother's serum showed the presence of very strong Rh blocking antibodies as well as a positive test with Rh + control cells suspended in normal serum. The titer of the blocking antibody increased from 1:4 to 1:64 during the fourth month of the pregnancy and remained at the higher level for more than two months after delivery of the dead fetus.

DISCUSSION

What is the prognosis for future pregnancies? As a serologist I am convinced that this husband and wife, because of their Rh relationship, can never have a normal baby, or, indeed, a living one. Unless or until a means of de-immunization is worked out this woman can have no living children by her husband. Thus one may contemplate the end result of a chain of patho-physiolgic events innocently started with a blood transfusion nearly nine years before the patient's first pregnancy. (Note—Certain aspects of this case have been previously discussed elsewhere.[10])

CASE NO. 2

During the winter of 1939-40 this patient, a young lady then 20 years old, was admitted to the hospital for treatment of a ruptured appendix. During her stay she was given a total of eight group compatible blood transfusions, with no untoward reactions. Subsequently she married, and in August, 1943, gave birth to a full term stillborn, macerated fetus. (Apparently no postmortem examination was done, as records were not available.)

In July, 1945, the patient was referred to me for Rh examination. She was then about three and a half months pregnant (grav. ii), the E. D. C. being January 29, 1946, and was found to be group B Rh—. Her serum contained weak anti-Rh agglutinins and moderately strong blocking antibody. Just how much of this was residual from her first pregnancy was difficult to decide. The husband was found to be Group O Rh +. These findings were confirmed by Dr. Levine, but the genotype of the husband could not be definitely established.

Because of the patient's history it was decided to check her serum at four to five week intervals. Relatively early the blocking antibody titer reached 1:128 and with one exception persisted at that level for all subsequent tests. The anti-Rh agglutinins increased slowly in strength during the course of the pregnancy while the slide agglutinin test increased noticeably in the latter part of the seventh month.

In view of the serologic findings the obstetrician decided to perform a cesarean section approximately one month before term, in an effort to remove the baby from its serologically harmful environment. The operation was performed uneventfully on December 29 and a six and a half pound, viable male infant delivered. The baby was not jaundiced. About 50 c.c. of group compatible Rh — blood were given in an umbilical vein immediately after birth. By the morning of December 30 the baby had become deeply jaundiced and the blood count had dropped to 3.5 million, with 60 per cent hemoglobin. The baby, being group B Rh+, was given a transfusion of 75 c.c. of Group B Rh— blood, with the blood count increasing to 4.6 million, and hemoglobin to 75 per cent. No erythroblasts were present in blood smears. However, there was marked hepatic enlargment and slight enlargement of the spleen. In spite of a third transfusion of 90 c.c. Group B Rh— blood, the baby failed rapidly and expired on the morning of December 31, about six hours after developing a pulmonary hemorrhage. One week following the delivery, the mother's serum showed the continued presence of strong Rh antibodies.

DISCUSSION

In this case one is again faced with the problem of giving the husband and wife a prognosis for future pregnancies. Because the husband's Rh genotype is in doubt, so that he might be heterozygous (Rhrh) and thereby capable of fathering an Rh—child, the prognosis is somewhat better than in the preceding case. However, even if one actually knew the father to be heterozygous, the prognosis could go no further actually than to state that while an Rh—child is possible in this couple and would not cause the mother to proliferate antibodies, there is no way of determining if the next child would be Rh+ or Rh—.

We were able to test the bloods of four of the people who donated blood to the patient in 1939-'40 and found one of them to be Group B Rh+ and three of them to be Group B Rh—. (One of the Rh—donors was the patient's brother.)

In 1939, when this girl was first a patient, if the knowledge of Rh had been comparable to its present development there is every reason to believe that her first child would have survived, for had we known of the importance of Rh typing young women, this patient would have received only Rh—blood and thus would not have been sensitized to the Rh factor prior to her first pregnancy.

CASE NO. 3

In December, 1939, the patient, then a girl 16 years old, was admitted to the hospital for treatment of first and second degree burns of the upper extremities. On December 31 she received, uneventfully, 380 c.c. of group compatible blood. Another transfusion, from a different donor, was given on January 2, 1940, with no untoward reaction.

On May 31, 1946, the patient, together with her husband and two year old daughter, was referred to the writer for Rh examination. The patient was found to be Group O, Rh—; the husband Group A, Rh+; and the little girl Group O, Rh+. Testing of the patient's serum revealed no definite evidence of anti-Rh antibodies, although there seemed to be a faint trace of blocking antibody present. No especial significance was attached to this since the patient was only six weeks pregnant at the time of her first visit to the laboratory. She was requested to return in about four weeks' time. On July 7 her blood showed definite blocking antibody, with a titer of 1:8 as well as other Rh antibodies in weaker strength. The question immediately arises as to when the patient was first sensitized to the Rh factor. One of the donors who gave her blood in 1939-40 was recently typed and found to be Rh+ and group compatible. Thus one (or perhaps both) of the transfusions could have initiated the isoimmunization, while her first pregnancy (with Rh+ baby) could have intensified it. Now she is pregnant again and it is predicted that the fetus is Rh+. There is no other explanation for the presence of Rh antibodies so early in the pregnancy except, perhaps, that they are residual from the first pregnancy. If they were present then they were of insufficient strength to injure the first child. At any rate, the patient is again pregnant, has antibodies and the prognosis is not good because the antibodies are of the blocking variety. Perhaps it is a moot question as to just how much the blood transfusions of six years ago contributed to the patient's present immunologic status. Most probably they initiated the sensitivity which has progressed to such a level that now in the third month of the second pregnancy the patient has sufficient blocking antibody present in her serum to cast grave doubts on the survival of the fetus.

DISCUSSION

It will be noted in all three of the above cases that the Rh—recipient was given Rh+ blood quite accidentally because in two cases the procedures were carried out prior to the discovery of the Rh complex and in the other not enough was known of the subject at the time. It is of course quite evident that such episodes, involving the administration of Rh+blood to an Rh—recipient, can (and still do) occur in spite of the tremendous amount of working information that we now have on the Rh factor. Such unfortunate occurrences may result either from failure to Rh type the blood of the patient or may lie in erroneous typing of the patient and/or donor. To my mind, failure to routinely Rh type transfusion patients (most especially females) is a greater sin than to mistype a blood once in a while. The latter will happen in the best of laboratories and the larger the service and the more personnel concerned, the more likely are such mistakes to occur. But, the very least that can be done is to try to establish accurate methods of Rh typing and then use them routinely. If a blood is occasionally Rh mistyped currently available procedures usually serve to detect the error before the blood is administered to the patient. Recently the writer had occasion to examine the blood of a patient concerned in just such an episode. The patient is a 58 year old white male who has cirrhosis of the liver, and in past months has been receiving frequent transfusions. Originally his blood was classified as Group A, Rh+, and in the course of several weeks he uneventfully received some eight pints of Rh +blood. A recheck was made on his Rh type and it was found actually to be Rh—. Since then he has received three pints of Rh—blood with no untoward reactions. A sample of his blood was sent to this laboratory to be examined for irregular agglutinins, particularly autoagglutinins. Actually no such agglutinins were present, but the serum did contain very potent anti-Rh agglutinins. The patient was found to be Group A_1 Rh—.

This case is mentioned at some length because it is an example of Rh sensitization in a male patient as a result of repeated transfusions of Rh+blood and because it characteristically illustrates that several pints of positive blood are usually required to sensitize an Rh—male. It should be noted that a single pregnancy often has more immunizing power than a large number of transfusions alone.

Cases such as those presented herein are admittedly rare, even as Rh isoimmunization itself is infrequent. Yet we cannot afford to overlook the fact that transfusion of Rh+blood at any age may produce a sensitivity in an Rh—person which will persist for years or perhaps for life, a potentially evil thing lurking in the reticulo-endothelial system and ready, as soon as the antigen (Rh+blood) is reintroduced into the body, to injure the patient, or, in the case of a woman, even destroy her children before they are born. Again, one must admit that such cases are statistically rare, compared with other medical conditions, but they are very real and poignant tragedies to the husbands and wives concerned. These cases can be prevented — they must be prevented.

SUPPLEMENT

Some additional remarks on this phase of the Rh problem are indicated by new information becoming available since the foregoing paper was written.

For one thing, we must abandon the idea that only about 5 per cent of Rh—people are capable of isoimmunization. Diamond[11] has shown that a more accurate estimate is nearer 50 per cent, or some ten times greater. It can be appreciated, therefore, that the potential danger inherent in disregarding the Rh factor in transfusions to females is proportionately greater than had been assumed.

Regarding male patients, I have had no occasion to change my earlier views, although Diamond's figure applies equally to them. As long as *ample* supplies of Rh—blood continue difficult to procure we do not propose routinely to Rh type males except, as stated above, when this procedure is indicated by the patient's transfusion history. It must be remembered that not every person in the 15 per cent Rh— segment of the general population is suitable as a donor, as this group includes children, old people and sick individuals as well as the Rh— patients requiring transfusion. Very probably not more than 5 per cent of the Rh— people in the average community are available as donors at any one time.

Another early concept that must be altered is that the Rh factor is confined to the erythrocytes and is not secreted in various body fluids as are the A and B factors.

In enlarging on an earlier report[12] Witebsky[13] recently commented on finding Rh substance in the amniotic fluid of some Rh+ fetuses and in the saliva and gastric juice of a few other Rh+ individuals. This may serve to explain some of the occasional normal Rh+ babies born to Rh— mothers who have exhibited antibodies during pregnancy. Further it brings nearer the realm of probability a preparation suitable for the *in vivo* neutralization of Rh antibodies. The problem of antigenicity of such a substance has been attacked by Calvin[14] and others.

Even if we had such an agent it would still behoove us to continue our efforts to prevent such cases as have been reported above. It is felt that a few good reasons, at least, have been advanced why the prevention of Rh isoimmunization in female patients is of vital importance. In conclusion, and without being overly dramatic, one may truthfully say that the transfusion of Rh+ blood into an Rh— girl or woman may constitute the death warrant of a child not only unborn but unconceived.*

Permission for the publication of these cases was very kindly granted by the following: Case No. 1, Dr. Edward L. King, Dr. Joseph W. Reddoch, and Dr. Thomas B. Sellers. Case No. 2, Dr. Edward L. King. Case No. 3, Dr. C. Gordon Johnson.

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*The grave prognosis given in case 3 has been justified by subsequent events. The patient exhibited increasingly strong antibodies and at seven months spontaneously delivered an Rh+ male infant who expired in thirty minutes. Postmortem examination revealed typical erythroblastosis fetalis, with hepatic and splenic enlargements, characteristic tissue changes and the presence of large numbers of immature erythrocytes in the peripheral circulation. The baby's cord blood contained Rh blocking antibody.

DIVERTICULOSIS OF THE COLON*

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Diverticulosis is a pathologic condition characterized by one or more diverticula in some portion of the intestinal tract. My paper is exclusively devoted to colon. These diverticula usually occur in multiple, more usually found segregated in some limited portion of the bowel and more frequently located in the sigmoid section of the left half of the large intestine. They are saccular pouches extending outwardly and lined with the mucous membrane of the bowel. For a long time they have been classified into two general types, the true and the false. In the former all the coats of the intestine are found, that is, the mucosa, muscularis, and the serosa; in the false type the muscularis is absent. Quite often the opening into these sacs is very small, which can easily induce retention of fecal material and subsequent inflammation. It has been found that in approximately 76 per cent of the cases the sigmoid flexure is the most common site of development and the most frequent site of diverticulitis. Most commonly the age of incidence is past 40, and the rate of incidence is 5 per cent. Especially prone are the older heavier persons who lead a sedentary life. Frequently these saccular pouches border along the longitudinal bands. The possible complicatory developments are:

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*Presented at the Staff Meeting of Our Lady of the Lake Sanitarium, July 24, 1946.

1. Diverticulitis.
2. Abscess formation.
3. Perforation with peritonitis.
4. Intestinal obstruction.
5. Internal or external fistula.
6. Carcinoma (descending order of incidence).

The symptomatology of diverticulosis without complications is characterized by constipation, chronic indigestion, flatulence, a recurrent dull tender ache in the lower half of the abdomen and a tenderness in the same area.

With inflammation there is pain in the involved side, fever, anorexia, tenderness and occasionally the passage of blood-stained mucus. A tumor mass may occasionally be found in the iliac fossa. These signs may be complemented with an abdominal rigidity of the involved half. However, not infrequently the abdomen may be soft and yielding to palpation. Not infrequently suggestive evidence of appendicitis may lead one to a hasty diagnosis of appendicitis.

When surgery becomes the elective approach a coloenterostomy is preferred. However, if the patient is very critically ill and surgical risk is at its maximum, a simple amputation of the diverticulum with plastic repair may be decided upon on laparotomy. If surgical risk encourages the surgeon a complete resection is done in behalf of therapeusis and prophylaxis. If surgery cannot be resorted to, the medical treatment for appendicitis may be rather efficacious.

I am especially interested in converging my interest in the particular complication of perforation with subsequent pelvic bacillus coli peritonitis. The following two cases will help to illustrate this phase of diverticulosis and convey the clinical picture for personal and professional estimate. These two cases are especially interesting because they ran a close parallel in mode of onset, progress and termination.

CASE NO. 1

Mrs. W. G., a white female, aged 82, was admitted to the hospital on May 7, 1946 and placed in bed for complete observation and treatment. This case, on admission, was categorized an acute medical emergency case.

She ate a hearty meal on May 6 at 10 a. m., and this was followed with pylorospasm and nausea with vomiting. Four hours later she developed a fever of 103 degrees. The following day, on May 7, prior to hospitalization, the patient went into shock at home, that is, she had a cardiovascular collapse. Her blood pressure dropped to 74, she was cold and clammy, in a semiconscious state and definitely in a very critical state. That evening I gave her two ampoules of coramine and then summoned an ambulance. She responded to emergency treatment and cardiovascular collapse was checked. The fever was rather high, but on the ninth day of May she definitely improved. Incidentally, streptomycin was begun at that time. My initial diagnosis on May 6 was Flexner gastroenteritis with cardiovascular collapse, but on May 7, I very quickly changed my diagnosis to sigmoid diverticulosis with perforation and subsequent bacillus coli pelvic peritonitis. During the course of this illness I became very much enthused over the fact that beginning with the ninth day of May, the patient began to improve and remained improved, but unfortunately in the morning of May 12, this patient very suddenly expired rather unexpectedly from possibly an embolus, a coronary thrombosis or an acidosis. On May 9, the patient had developed an uremia of N.P.N. 100, urea 50, and creatinine 2 but with the intravenous use of 20 per cent glucose, theophylline, salyrgan and digifoline the uremia improved to N.P.N. 63, urea 32, and creatinine 1.2 on May 11. Also the urinary output during these days improved from 8 ounces to 42 ounces. Blood count (5-8-46) white blood cells 12,850, SL 36, LM 2, polys 62. On May 9, hemoglobin 75, red blood cells 4,320,000, white blood cells 14,150, SL 28, LM 2, polys 69, E 1, no plasmodia found.

The sedimentation rate was: 15 min. 20, 30 min. 29, 60 min. 36.

Urinalysis on May 8 and 9 was negative.

Treatment: (emergency measures.) May 7:

1. Saline—10 per cent glucose, one quart.
2. Coramine—Two ampoules, intravenous infusion.
3. Digifoline—Two ampoules intravenously. One hypodermic every two hours for six doses.
4. Elevate foot of bed one foot.
5. Shock measures—heat.
   (B. P. has risen from 76 to 140 systolic).

Routine Orders:

1. Digifoline ampoule, one hypodermic every four hours for eight doses.
2. Coramine ampoule hypodermic whenever necessary, if in shock and pulse weak.

3. Saline 10 per cent—Glucose one quart infusion daily.
4. Diet.
    1. Nothing ice cold.
    2. No soft drinks.
    3. No ice cream.
    4. No fruit juices.
    5. No grease or fried foods.
    6. No gravies.

    May have:
    1. Boiled milk served warm.
    2. Mashed potatoes—cream of wheat.
    3. Grits—oatmeal.
    4. Custards.
    5. Warm tea, coffee, cocoa, milk.
    6. Cool water.
5. Penicillin, 20,000 U. every three hours for sixteen doses.
6. Sulfadiazine, grs. 7½ tab., one every three hours, six doses daily.
7. Aspirin, grs. 5—whenever necessary for fever if high.
8. $NaHCO_3$ drahms ½, six times a day.
9. Catheterize p. r. n.
10. Sodium luminal, grs. 3, whenever necessary.

May 8, 1946:

1. Complete blood count repeated.
2. Sed rate.
3. N. P. N.
4. Catheterized specimen of urine to laboratory.
5. Continue digifoline, one ampoule every four hours.
6. Nothing by mouth.
7. No enemas.
8. Streptomycin has been ordered.

May 9, 1946:

1. Streptomycin every three hours.
2. Discontinue penicillin.

May 10, 1946:

1. Digifoline one ampoule, twice daily.
2. Salyrgan-theophylline intravenously.
3. Glucose 50 per cent intravenously.
4. Coramine—intravenously.
5. N. P. N. and creatinine.
6. Repeat blood count.

May 11, 1946:

1. Diet starting in a. m.:
    1. Grapefruit juice.
    2. Sweet lemonade.
    3. Coffee.
    4. Broth.
    5. Creamed potatoes.
2. Place under oxygen tent.
3. Upjohn's adrenal extract hypodermic 2 c.c. every four hours.
4. Amino acids parenteral (intravenously) Stearns.

CASE NO. 2

Mrs. E. F., a white female, age 60, was admitted to the hospital on August 30, 1944, per ambulance and placed in bed for complete observation and treatment. This case, on admission, was categorized an acute medical emergency case. In the early morning at 4 o'clock she was aroused with much pain in the lower abdomen with immediate nausea and emesis in about 20 minutes. She went to the kitchen for a cup of coffee, but had another spell of pain and vomited immediately after. She improved, however, sufficiently to return to bed and finish her night's sleep. During the early part of the day she did not feel quite well, so a doctor was summoned and at 8 p. m. she was admitted to the hospital. The patient was moderately tall, rather slender, somewhat emaciated, not apparently very ill on superficial observation, but revealing marked tenderness in the lower left quadrant of the abdomen along the left flank.

Patient stated that she had observed the passage of a black stool 24 hours prior to onset of present illness.

History: Present illness as revealed in preliminary resume.

Past History: Eight months ago she had an intestinal upset which responded to medical treatment, tenderness in lower abdomen with diarrhea, a spell of pleurisy in 1934, arthritis for a few years, appendectomy in 1935, no accidents, usual childhood diseases, no history of amebiasis or typhoid fever.

Family History: No history of cancer or tuberculosis in the family.

Physical Examination: General appearance: a white female definitely senile, impression age 65 or 70, well developed, but very poorly nourished, marginally emaciated, complexion not clear and somewhat sallow, dehydrated and run down. Head essentially negative, except for prominent tortuosity of the right temple artery, face wrinkled, skin soft and moist. E. E. N. T.: Essentially negative. Chest: Lung field clear. Abdomen: Right rectus incisional scar present and definite tenderness in left lumbar region radiating to the left iliac region (peristalsis normal, no palpable masses). Extremities: Negative. Vaginal: atrophic pelvic organs, tenderness in lower left quadrant and suggestion of a mass in that region. Rectal: Findings same as vaginal. Heart: (Chest) An occasional irregularity of rhythm, no murmurs, heart slightly small.

Progress: Patient did not improve, continued growing worse from day to day, and expired on September 18, 1944 at 6 a. m. Patient, during course of illness, developed a hypostatic pneumonitis and cardiac failure.

Laboratory Studies: August 30, 1944: white blood cells 19,850, 79 polys, L 20, M 1. Malarial smear, negative (thin smear-Wright stain). Urinalysis: Sp. 10.20; albumin heavy trace; sugar, free; crystals, none; casts, few hyaline; epith. moderate amount; white blood cells, occasional; red blood cells, none. Blood Wassermann negative; Kolmer and Kahn, negative.

September 4, 1944: White blood cells 19,900, polys 73, L 25, M 2, red blood cells 8,000,000, hemoglobin 65.

Sed. rate: 15M-5M
30M-22M
60M-36M

Blood Chemistry 100 mg/cc.

N. P. N. 37.7 mg/cc; urea N 18 mg/cc; creatinine 1.3 mg/cc.

Urinalysis: Color, yellow; reaction, acid; sp. gr. 10.24; albumin faint trace; sugar free; indican, negative, acetone, negative; diacetic, negative; white blood cells occasional; red blood cells none; sulfadiazine crystals.

Differential diagnosis: Acute perforation posterior wall of the stomach with abscess of lesser peritoneal sac.

Perinephritic abscess left.

Carcinoma (G. I.).

Aneurysm (retroperitoneal).

Diverticulitis (sigmoid).

CONCLUSION

These two cases differ only in their duration. Case 2 survived for over three weeks and died rather suddenly, but never did get better. Case 1 survived six days and had actually improved, but unfortunately, she died from apparently an embolus, a thrombosis or an acidosis. In either case they did not develop an abdominal rigidity at any time during the course of the illness, but they both experienced a tenderness in the left iliac region where a palpable mass developed in the second day of their illness. The predominant features were: (1) Suddenness of onset; (2) shock; (3) fever; (4) pain and tenderness in lower abdomen to the left; (5) palpable mass; (6) no bowel movement; (7) nausea without vomiting; (8) general depression; (9) ashen-grey complexion; (10) vomiting following ingestion of a meal; (11) symptoms of gastroenteritis.

The one important thing about these cases which should be cited as a word of caution is that case 1 took a purgative. Case 2 had several enemas. These two things should definitely be placed on the prohibitive list. In conclusion I wish to say, for sake of emphasis, that any person, especially past 50, who is suddenly taken ill with symptoms of gastroenteritis complicated with toxicity, shock or semi-consciousness should be categorized into this particular group in the differential diagnosis.

AMNIOTOME

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The fetal membranes may be artificially ruptured to induce labor, to expedite the progress of labor, or to obtain the tamponade effect of the head in certain cases of placenta praevia. Obviously, rupture of the membranes should not be performed for the induction of labor unless there is a definite reason for terminating the pregnancy. If such an indication exists, then one must take into consideration the following: fetal size, presentation, position, station and condition; and the status of the cervix plus other maternal factors. The cervix should be soft, and its canal should permit the introduction of two fingers. Evidence of effacement is also a desirable indication of the "ripeness" of the situation.

Most obstetricians now agree that the advancing head is a better dilator than the hydrostatic bag which was so forcefully emphasized years ago. It is the general concensus that the distressful, long dry labors of the past were not the results of spontaneous early rupture of the membranes, but were due to faulty presentation, malposition, disproportion or other abnormalities.

On the Tulane Obstetrical Service at the New Orleans Charity Hospital, we have used various and sundry instruments to rupture fetal membranes before or after the onset of labor, but many of us have preferred to use one-half of a tenaculum.

Before the days of the box lock, it was easy enough to find a tenaculum which either had been discarded or was in such a condition that it was a candidate for the scrap pile. During recent years, however, such has not been the case. Therefore, it was advisable to design an amniotome* after the desirable features of our long-used and highly regarded "membrane hook." The result is shown in the figure,

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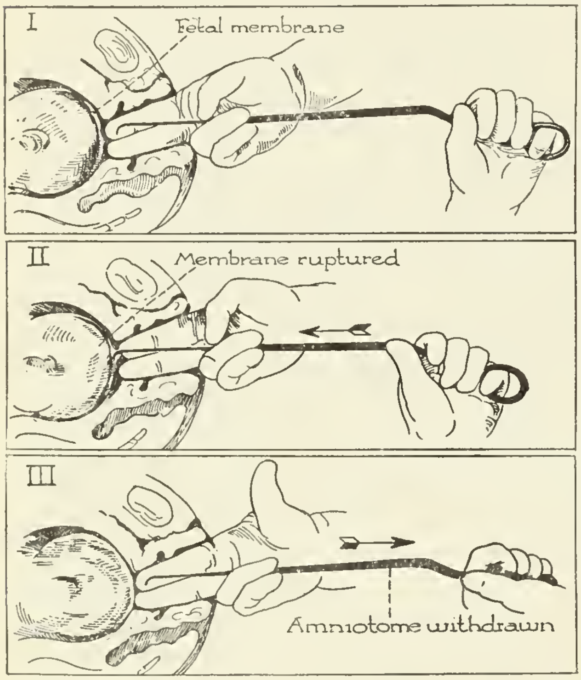
the actual length of the stainless steel instrument being nine inches.



It will be noted that in maneuver I, the finger guards the tip of the amniotome. In maneuver II, the amniotome is brought into contact with the fetal membranes and manipulated in such a way that perforation of the membranes is effected. Maneuver III indicates how the amniotome is withdrawn so that the tip of the index finger again guards against possible trauma to any of the maternal structures. As shown, the fingers should be left in position until fluid ceases to flow at a rapid rate, rather than be withdrawn with the first gush of water. After use, the instrument should be cleansed with soap and water and then sterilized. For sterilization we are now employing 1:1000 Zephiran solution.

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*The instrument may be obtained from the J. Sklar Manufacturing Company, 38-04 Woodside Avenue, Long Island City 4, New York.

THE TREATMENT OF GAMBIAN TRYPANOSOMIASIS*

JOHN K. MILLER, M. D.
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INTRODUCTION

In 1892 Laveran was able within the compass of a short article in the Archives de Médicine Expérimentale et d'Anatomie Pathologique to give a complete resume of all that was known concerning the trypanosomes. Only one pathogenic species was known and few people were interested in it because it produced only an obscure disease among livestock in India.

African sleeping sickness was described a hundred and forty-three years ago by Winterbottom in Sierra Leone but it was not until December 18, 1901 that Dutton and Ford recognized definitely for the first time that a trypanosome might occur in man and named this parasite *Trypanosoma gambiense.* Numerous bacteria, malnutrition, manioc poisoning and even a protozoan, *Filaria perstans,* had been blamed as the cause of sleeping sickness before this time. In 1903, after twelve more cases of human trypanosomiasis had been recorded, Castellani, examining the cerebrospinal fluid of natives with sleeping sickness in Uganda, discovered trypanosomes which were first called *Trypanosoma ugandense* (Castellani) and *Trypanosoma castellani* (Kruse). This observation was soon widely confirmed throughout Uganda and the Congo Free State.

It has since been shown that these two organisms are the same. A subspecies of *T. gambiense* which is morphologically indistinguishable from it but which causes a more severe human reaction and which is resistant to arsenic therapy, was described in 1910 by Stephens and Fantham and named *T. rhodesiense.* They considered this organism a distinct species but different authors have suggested that it may be a variant of *T. brucei* or *T. gambiense. T. rhodesiense* is usually transmitted by *Glossina morsitans* while *G. palpalis* is the most common insect vector for *T. gambiense.* The conclusion of the International Commission on Human Trypanosomiasis was that the two trypanosomes were the same organism transmitted by different vectors.

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*Awarded the Walter Reed Memorial Medal offered by the Louisiana State Medical Society for the best thesis on tropical or preventive medicine to a member of the senior class at Tulane Medical School.

EARLY MEANS OF THERAPY

It is interesting to read in Nabarro's translation of Laveran and Mesnil's treatise on trypanosomiasis (1907) of the early efforts at treatment of African sleeping sickness. The initial statement is: "Authors are agreed that sleeping sickness is always fatal. By careful nursing so that bedsores are prevented and the patients properly fed, one may prolong the life of patients, without, however, being able to prevent the fatal issue. Whether the prognosis is as grave in the first stage of the disease when the trypanosomes are not found in the cerebrospinal fluid, is at present undecided."

These authors recommended tonic treatment with iron, quinine and arsenic in the form of arsenious oxide of sodium arsenite. The discovery of the curative value of trypan red (Ehrlich and Shiga) upon caderas in mice had given great impetus to the study of the benzidine dyes as trypanocides. Atoxyl, described by Thomas in 1905, was showing great promise, especially in conjunction with trypan red or strychnine sulfate. The dose of atoxyl recommended varied with different authors from 0.6 to 1.5 gram.

In view of later discoveries it is interesting to note in more detail the work of Mesnil and Nicolle on the derivatives of benzidine. Numerous compounds were prepared and tested on various animals infected with *T. brucei, T. equinum, T. evansi, T. dimorpha,* and *T. gambiense.* Tolidine and dichlorobenzidine combined with 1, 8, aminonaphthol, 3, 6, disulphonic acid were the most effective in the first three infections. The two most effective compounds tested on *T. gambiense* were p-diamino-diphenylurea combined with 1, 8, aminonaphthol, 3, 6, disulphonic acid and p-amino phenylglycolether combined with the same acid. Although these dyes were not satisfactory trypanocides one notes how close these authors came to stumbling onto the diamidines which are receiving so much attention today.

Manson's manual of tropical diseases (fifth edition) published in 1914 was somewhat more optimistic than Laveran and Mesnil's work. Manson states: "Although something may be done to relieve symptoms and delay the fatal issue in the sleeping sickness stage of trypanosomiasis, it is only in the earlier stages of the infection and by the persistent and energetic use of arsenic or antimony, or both, that a cure can be effected, and that in a proportion of cases only."

The drugs which were in use at this time (1914) were atoxyl (soamine), arsenophenylglycin, arsacetin, and salvarsan. Manson recommended atoxyl given grains 3, intramuscularly every three days for two years in combination with tartar emetic. These and all other drugs tried, however, were found to be impotent in *T. rhodesiense* infections.

Atoxyl was the first really effective trypanocide discovered. Ouzilleau and Lefrou using atoxyl in doses of 1.0 to 1.25 gram combined with tartar emetic 0.05 to 0.1 gram reported a very favorable series. They gave a total of six injections over a twelve week period to 186 patients in the first or second class and had only 17 relapses after observation for over a year in 164 cases and over seven months in the remainder.

Contrary to the meaning of its name, atoxyl is extremely toxic. It combines the general toxic properties of all organic arsenicals with a special propensity for causing optic nerve injury. It is no longer employed in antiluetic therapy and is only rarely used in tryparsamide resistant cases in the treatment of trypanosomiasis.

Drug-fastness is easily produced with

atoxyl and Fulton and Yorke have demonstrated the permanence of the resistance by maintaining an atoxyl-fast strain for over 12 years with passage through 1528 mice.

TRYPARSAMIDE

Tryparsamide (sodium salt of N-phenylgycineamide-p-arsonic acid) was first prepared (1915) and described by Jacobs and Heidelberger in 1919. All of the early experimental work was done by Pearce and Brown in a manner which may be considered a classic in experimental therapeutics. The new drug was thoroughly and extensively tried out in *Trypanosoma brucei, T. gambiense, T. equinum, T. equiperdum*, and *T. evansi* infections in rabbits, rats, mice and guinea pigs, the therapeutic and lethal doses determined and the toxic symptoms and pathology were shown before the results were publicized. The efficacy of different methods of administration was tested and the intravenous route was found to be the best. Doses of over 0.3 gram per kilogram were found to be almost uniformly curative in all cases even when only one dose was given, and a single dose of only half this amount was highly effective. Special note was made that the drug seemed to have an excellent effect as a general tonic for animals in poor condition. Recovery from near lethal doses was rapid and complete and upon repeated administration the animals appeared to develop tolerance to the drug. Toxic reactions noted were tremors, clonic spasms, weakness, prostration, tics, anorexia, diarrhea, and weight loss. The pathology consisted of petechiae, effusions, cellular degeneration or necrosis of the kidneys, adrenals, cardiovascular system, gastrointestinal system, central nervous system and the blood and blood-forming organs.

In 1920 Dr. Pearce went to Leopoldville, Belgian Congo, where, assisted by Drs. Rodhain, Van den Branden and Van Hoof, she treated 77 cases of human infection with *T. gambiense*. She found that a single dose of 0.3 to 7.0 grams of tryparsamide produced sterilization of the blood in three and a half to 24 hours and with dosages of 0.5 to 5.0 grams the sterilization lasted three to six weeks. General physical condition was promptly improved, the subjective symptoms disappeared and temperature and pulse returned to normal in two to three days. Within a month the blood picture improved, the adenitis subsided and there was a gain in weight. Visual disturbances of a transitory nature were noted in nine of the 28 advanced cases. A number of relapses occurred during the brief period of observation but the results especially among those cases with central nervous system involvement were most heartening. A dose of 2 to 3 grams once weekly was recommended but the ideal total dose was still to be determined.

Chesterman in 1924 reported a series of 38 cases which had received 10 to 28 grams of tryparsamide and had been followed for one and a half to two years. Of 24 previously untreated cases nine were cured while of 10 who had previously received other arsenicals only three were well. The other four cases had been treated unsuccessfully by Marshall's serum method and three of these remained well after tryparsamide therapy. One case of blindness was reported.

In 1927 Kellersberger, Marugo, Lopes, Infante, and Van den Branden, reported 339 cases treated with tryparsamide of whom 220 were apparently cured. Many of these showed moderate or slight alteration of the spinal fluid. Trolli reviewed 1400 cases treated in the Belgian Congo prior to 1926 and stated that cure rates of 28 to 100 per cent had been obtained in the 896 cases with spinal fluid changes. Almost all of these recoveries were a true gain when compared with older forms of therapy which rarely cured an advanced case. Various dosages were recommended from 1.5 to 4 grams per dose once weekly to a total of 20 to 100 grams. Van den Branden reported a cure rate of over 17 per cent in late cases while Lauterberg stated that 60 per cent cures should be expected even in severest cases.

The most comprehensive survey of tryparsamide therapy to date is the monograph of the Rochefeller Institute by Louise Pearce published in August 1930. She referred to 1848 cases and analyzed 1197 of these; 1101 were advanced in degree and 96 were early cases. She urged the administration of the drug in weekly doses of 0.05 to 0.06 gram per kilogram. Early cases should receive a total of 0.4 to 0.5 gram per kilogram. Advanced cases were best treated by graduated doses beginning with 0.02 to 0.05 gram per kilogram and gradually working up to a full dose of 3 to 4 grams. A first course of 25 to 30 grams was suggested for these advanced cases, to be repeated after several months and again if needed. The incidence of visual disturbances was 5 per cent, of permanent impairment of vision 1.8 per cent. The results which she reported following five to 31 months' observation were as follows:

No. cases	Apparent cures	Improved	Failures
96 (early)	89 (92.7%)	0	7 (7.3%)
1101 (advanced)	646 (58.7%)	272 (24.7%)	183 (16.6%)

Kellersberger, who has given 150,000 injections of tryparsamide for sleeping sickness, considers it the most useful drug in this disease. He advocates average weekly doses of 0.045 gram per kilogram and states that in order to be fully effective the drug must be pushed to the limit of safety. Low doses such as 2 grams routinely to adults or graduated doses are likely to produce arsenoresistant trypanosomes.

Hawking in 1940 ran a number of experiments comparing the plasma and cerebrospinal fluid levels of tryparsamide and various other trypanocidal agents (neocryl, undecaine diamidine, neoarsphenamine). He concluded that the ability of tryparsamide to penetrate the blood-spinal fluid barrier did not depend on the presence of trypanosomal lesions in the choroid plexus as other authors had suggested. The amount of tryparsamide in the spinal fluid (41 gamma of total arsenic at 17 hours), however, was insufficient to affect freshly isolated trypanosomes.

Fowler treated 42 cases of Gambian sleeping sickness by an intravenous drip giving 2 grams of tryparsamide daily for six to nine days. A rest was often necessary as the patients became exhausted with the high fever produced. In three patients visual acuity was almost lost; seven patients died from the treatment. The 35 survivors were clinically cured and four to seven months later 24 were reexamined and found to be well. In 33 of the 42 cases there was spinal fluid involvement.

Most authors have found that the maximum cure rate with the minimum number of accidents may be secured by using what is called the combined form of treatment. This consists of one or more 1 gram doses of Bayer 205 followed by a number of doses of tryparsamide. Harding and Lourie recommend one to three doses of Bayer 205 at weekly intervals followed by a rest of five to seven weeks and then by three to nine or more doses of tryparsamide. This method tends greatly to reduce toxic reactions since the kidney is allowed to recover before the arsenical is begun.

As has been mentioned before the chief toxic reaction to tryparsamide is the visual injury. Ideally all persons to be treated should have tests run to determine their visual fields and acuity. If any impairment is present great care should be exercised. Ordinarily close questioning during treatment will detect any visual disturbances before permanent harm is done. Most authors agree that these reactions almost always develop during the first eight to 12 injections or not at all. The incidence is reported as 5 to 10 per cent by various writers but only a small portion of these cases show permanent harm. In some cases sudden blindness may occur. The subjective complaints are usually blurring, haziness, spots or clouds before the eyes, shimmering, dazzling or blindness. Objectively narrowing of the visual fields or loss of visual acuity may be demonstrable.

Other untoward reactions include nitritoid reactions; mild jaundice, or very rarely liver atrophy; gastrointestinal disorders such as nausea, vomiting, diarrhea and abdominal cramps; desquamative dermatitis and urticaria. Cerebral symptoms may include dizziness, headaches, drowsiness, coma, convulsions, siezures, tremors, tingling sensations, acute hallucinosis, foot drop, or emotional disturbances.

The possibility of the development of tryparsamide resistant trypanosomes was suspected as soon as the drug was discovered since it was well known that trypanosomes and spirochaetes readily became resistant to atoxyl and other arsenicals. This has been amply confirmed and De Brauwere has reported the development of a number of arsenic resistant foci of trypanosomiasis in the Belgian Congo. Yorke was able to maintain strains of *T. gambiense* for many years after they had been experimentally made arseno-resistant without appreciable loss of this characteristic. King suggests several modes of action of the arsonic acid group on the trypanosomes. Nonionized compounds may orient themselves at a water-lipoid interface while others may be absorbed on polar surfaces. The killing reaction probably involves the arsenoxide group and the mercaptan groups of the living cells.

BAYER 205

In 1920 Haendel and Joetten published an article dealing with a new Bayer preparation whose formula was kept secret on account of the position of German industry at that time. This drug, known as Bayer 205, showed a remarkable trypanocidal and prophylactic action in mice infected with *T. equiperdum*, *T. congolense*, and *T. brucei* and also in rabbits, rats, guinea pigs and dogs infected with various trypanosomes.

The composition of the drug has never been divulged by the manufacturers but substances apparently identical have been synthesized by other companies (Fourneau 309, Moranyl, Antrypol, etc.). It is a sulfonated naphthalene amidopolypeptide (see appendix for formula) with a relatively low toxicity. Large doses cause hemolysis and decreased coagulability of the blood and result in hemorrhages and nephritis. Degenerative lesions of the adrenal cortex were produced by Humphreys in animals with a dose one and a half to two times the maximum safe human dose (0.02 gram per kilogram).

Bayer 205 has little effect on trypanosomes *in vitro* but the serum of animals or humans which have received even one dose retains a trypanocidal action for weeks or months when injected into other infected animals. The sterilization of the blood is rapid in mice and slower in other animals. Various ideas have been suggested to explain the mode of action of this drug. Mayer and Zeiss after noticing that the trypanosomes were seen in dividing forms frequently after treating animals with Bayer 205 suggested that there was some interference or blockage of reproduction of the flagellates. Jasco and Hawking explained the action of the drug as an opsonizing effect but Dubois and Kohn were able to disprove this. These authors treated infected mice with moderate doses of Bayer 205 and 12 to 14 hours later injected blood from these animals into other uninfected mice whose reticuloendothelial systems had been blocked by electrocolloidal copper and splenectomy. These mice did not become infected so the authors concluded that the trypanosomes themselves had fixed a toxic amount of the dye.

The trypanocidal activity of the serum from healthy or cured animals or humans treated with Bayer 205 has received a good deal of attention. Boursnell and Wormall have shown that the drug is retained chiefly by the kidneys, spleen and plasma globulins apparently by a chemical combination. The serum proteins undergo a marked change on the addition of 2 per cent Bayer 205 solution. They become incoagulable by tannic acid, metallic salts or heating to 60° C. and fail to provoke an anaphylactic shock in sensitized animals due to a denaturation in which the sulfonic group is probably involved. A complex combination with serum calcium is probably concerned in the anticoagulant effect. Hawking was able to demonstrate a measurable amount of Bayer 205 in the plasma of individuals who had received eight to nine 1 gram doses eight to nine months previously. He also reported that there is a great individual variation in the plasma level produced in different persons by the same dosage. He found that the cerebrospinal fluid levels were too low to measure even when the plasma level reached 15 mg. per cent. On the other hand, Kunert, in studying the blood and cerebrospinal fluid concluded that the blood level was two to four times as high as that in the spinal fluid and that the drug reached the spinal fluid by either means of administration.

The early reports on the efficacy of Bayer 205 were often enthusiastic. Kellersberger reported nearly 100 per cent cure in early cases treated with 5 to 10 grams. Kleine and Fischer after having poor results in 30 late cases treated 163 early with three 1 gram doses and found only two relapses among 90 of them six months later. Most of the workers reported excellent results in early cases but only fair or poor results after the nervous system had become involved. Strada and Lopes, however, treated 12 early cases and 31 late cases and obtained only one cure.

More recent reports have uniformly agreed that Bayer 205 should never be relied upon in late trypanosomiasis. Saunders followed 35 late cases for 13 years and at the end of that time 34 had died of sleeping sickness and the thirty-fifth had relapsed. He states that Bayer 205 will not cure central nervous system involvement but may produce remissions lasting for years. This fact is of value in the treatment of late cases which are arsenic resistant.

For the treatment of early trypanosomiasis Bayer 205 is recommended because of its low toxicity and high index of effectiveness. The average recommended dose for intravenous administration is 1 gram dissolved in 10 c. c. of distilled water. The total amount required to effect a cure is about 10 grams. Intramuscular injection is sometimes used. Intrathecal administration is to be condemned as it does not improve the effectiveness and produces pain, vomiting, headache, convulsions, and even death.

Bayer 205 is not, however, without toxic danger. A temporary nephritis evidenced by albuminuria is often produced and may terminate fatally. Other toxic reactions which have been noted are nausea, vomiting, fever over 40° C., palpebral edema, cutaneous hyperesthesia, photophobia, epistaxis, dermatitis, stomatitis, conjunctivitis, and urticaria. Idiosyncrasy to the drug with alarming collapse and one death was reported by Edge. He advocates a trial dose of 0.3 gram to avoid such misfortunes.

Hawking described three cases which relapsed five, nine, and 19 days after the last dose of Bayer 205. Chemical determination of the blood levels of the drug showed that there had been defective accumulation. The trypanosomes in two of these cases were proved to be non-resistant to Bayer 205.

Drug fastness to Bayer 205 has been demonstrated but different workers have gotten different results in determining the persistence of this resistance. Fulton and Yorke maintained a resistant strain of trypanosomes for over three and a half years without loss of fastness.

In the field of prophylaxis of sleeping sickness Bayer 205 has proved its greatest value. Van den Branden in 1927 gave two 1 gram injections to over 183 natives in a village in the Belgian Congo. All of these people had received semiannual examinations for the previous five years so the new infection rate was well established. Following the experiment the number of new infections dropped from 17 to three. Fain in 1943 administered 0.025 gram per kilogram to 4500 persons in an old focus of sleeping sickness and reduced the new case rate from 162 to 12. Other workers have obtained similar results. Van Hoof and his coworkers recommended a dose of 1.25 to 1.5 gram to adults repeated after three months. They strongly urge that persons so treated should be carefully examined before treatment and when giving the second dose to avoid masking an infection which is already established.

DIAMIDINE COMPOUNDS

The story of the discovery of the effectiveness of this important group of drugs is vividly portrayed by the address given by Professor Warrington Yorke before the Royal Society of Tropical Medicine and Hygiene. In 1911 Biot, Biot, Richard and Fleig showed that if glucose was added to citrated blood containing trypanosomes the survival time of the organisms was prolonged. Later authors further investigated this phenomenon proving that enormous quantities of glucose were required in the metabolism of trypanosomes and that animals infected with these organisms exhibited varying degrees of hypoglycemia during the course of the infection.

Jansco and Jansco in 1935, having reached the conclusion that Bayer 205 acts by interfering with the carbohydrate metabolism of the trypanosomes, examined a number of guanidine derivatives, which were known to produce hypoglycemia, in the treatment of experimental trypanosomiasis. They found that a number of these compounds, notably synthalin, showed a therapeutic action against *T. brucei.* In the same year Schern and Artagaveytia-Allende concluded that the therapeutic action of synthalin was due to a toxic effect on the trypanosomes (*T. equinum*) as well as to the hypoglycemia produced. Proceeding further along this line in 1939 Lourie and Yorke proved that the therapeutic effect was not due to the hypoglycemia at all since the small doses employed had no significant effect on the blood sugar level and secondly that insulin had no trypanocidal effect. These authors, having developed a method for maintaining trypanosomes *in vitro,* went on to show that synthalin has a powerful, direct trypanocidal effect. Thus we see that after starting with a false hypothesis a really therapeutically active drug was discovered.

This discovery was of especial importance because synthalin did not resemble structurally any of the known trypanocidal substances.

Investigations were carried out by the National Institute for Medical Research and later by the drug firm, May and Baker of London, to determine what portions of the chemical structure of synthalin contained the trypanocidal activity. Numerous compounds were tested and several were eventually selected which showed very high trypanocidal activity. These were:

1. Undecaine-1, 11-diamidine
2. 4:4'diamidino diphenyl ether (phenamidine)
3. 4:4'diamidino stilbene (stilbamidine)
4. 4:4'diamidino diphenoxy propane (propamidine)
5. 4:4'diamidino diphenoxy pentane (pentamidine)

The last three compounds as well as a number of others which exhibited less trypanocidal activity have in common an inert carbon chain joined directly or through oxygen atoms to two diamidino groups. Relatively small changes in the polar groups such as changing them to a 3:4' or 3:3' position or replacement of one hydrogen by a methyl group completely destroy the trypanocidal activity.

Little progress has been made in determining the method of action of this group of compounds. One of the main difficulties is that no entirely satisfactory method has yet been devised for chemically estimating the absorption and excretion of the drugs. Hawking and others have made use of the blue fluorescence produced by 4:4'diamidino stilbene in ultraviolet light to show that it is absorbed by living trypanosomes, deposited in the liver, kidneys, and skin and excreted in the urine. The blood plasma level drops rapidly following injection into infected animals. A colorimetric method of analysis has also been described by Devine.

The metabolic effects of the 4:4'diamidino derivatives of diphenyl ether, stilbene, diphenoxy propane and of diphenoxy pentane have been carefully studied by Devine and by Wein and his coworkers. They reported that the diphenyl ether compound was slightly less toxic than the other three. The effects produced were a drop in blood pressure caused by peripheral vasodilatation, and a stimulation of smooth muscle. The first effect could be reduced by the administration of calcium or atropine but atropine did not counteract the smooth muscle stimulation. An ergotoxin-like effect was noted and the actions of adrenalin or the blood pressure, uterus and perfused vessels were reduced. Dosages on the therapeutic order of these drugs produced no appreciable change in the blood picture or in the chemical constituents of the blood. Higher doses, some approaching the toxic level, produced hyperglycemia, raised blood urea and reduced serum calcium and potassium. Repeated high doses produced marked depletion of liver glycogen, fatty degeneration of the liver and cloudy swelling of the kidneys.

Following a report by Bowesman which seemed to indicate that old solutions of 4:4'diamidino stilbene showed increased toxicity Fulton and Yorke and other authors began to study this phenomenon. They found that the saturated diamidines were quite stable under all conditions and that solutions of the stilbene compound would withstand heat or storage in the dark but rapidly became more toxic when exposed to sunlight. Henry reports that this change occurs largely during the first six hours of exposure to a mercury arc lamp or to intense tropical sunlight. Several explanations have been offered. Barber, Slack and Wein believe that the toxicity is due to 4:4'diamidino phenyl benzyl carbinol produced by the addition to the unsaturated stilbene chain of water elements. Henry on the other hand is convinced that the amidine groups are changed into amido groups and possibly that polymerization occurs forming 1, 2, 3, 4-tetra (4'amidino-phenyl) cyclobutane. This point is yet to be settled and meanwhile the use of freshly prepared solutions of 4:4'diamidino stilbene will prevent the toxic reactions from occurring.

The clinical use of the diamidines has been limited almost entirely to three compounds—stilbamidine, pentamidine and propamidine.

McLetchie (Nigeria 1940) reported a small series of 14 cases treated with stilbamide. In eight mild cases he found that an average dose of 8.8 mg. per kilogram given intravenously over a nine day period was as effective as Bayer 205 but less so than the combined treatment. The short course of therapy is a great advantage over the older methods. Harding (Nigeria 1940) in a similar study of 13 cases agreed with McLetchie that only early cases responded favorably to this drug. He reported that in no cases with pathological spinal fluid was the drug effective. The dosage which he used varied in the cured cases from 3.2 to 8.0 mg. per kilogram. Bowesman (Gambia 1940) treated 36 cases with seven to ten doses of 1 mg. per kilogram. He agreed that in early cases the drug is effective and places the criterion that the cerebrospinal fluid protein must be below 0.05 per cent. He concludes that stilbamidine is the drug of choice in early trypanosomiasis because of its low toxicity, relatively high effectiveness and the short period required for its administration. Lourie (Sierra Leone 1942) reported 153 cases treated with stilbamidine. Of the 136 which were not far advanced 128 were traced for over a year and 51 of these were well. The dosage was one to eight doses of 50 mg. The author concluded that this drug is less effective than either propamidine, pentamidine, or tryparsamide even in early cases.

Pentamidine has been used on a fairly large number of patients. Saunders reported a high cure rate (11 out of 12) in cases with less than 30 cells per cu. mm. in the spinal fluid. About half of 35 cases with a higher cell count were improved but relapses were common. He recommended a dosage of 2 mg. per kilogram of body weight. Harding reported excellent results in 192 cases using eight to 12 daily doses of 50 to 100 mg. He considers this the drug of choice for cases with a spinal fluid cell count below 10 to 15 per cu. mm. Lawson and Gilbert after reporting series of 53 and 14 cases respectively also urge the use of this drug in early cases because of its low toxicity and effectiveness. The maximum dosage which may be given without toxic manifestations is 2 mg. per kilogram twice weekly since the drug tends to accumulate.

Van Hoof, Henrard, and Peel found that a single dose of 2 to 3 mg. of pentamidine per kilogram produced a prophylaxis lasting 10 to 12 months in two volunteers. Fulton using mice was able to demonstrate a prophylactic effect lasting up to four weeks.

The toxic reactions to the diamidine

drugs may be divided in two groups; those which occur immediately and those which are delayed. The former are quite common, the latter relatively rare. Immediate effects are usually those associated with a sudden fall in blood pressure—rapid, thin or imperceptible pulse, sweating, dizziness, faintness, unconsciousness, epileptiform twitchings and rarely incontinence are seen. Other effects may include salivation, nausea, vomiting, a puffy suffusion of the face and eyelids, severe pruritis, headache, rigor, palpitations, a sense of pressure in the head, albuminuria, and leukopenia. Lourie states that these effects were so severe following the use of stilbamidine that sooner or later an immediate fatal collapse is to be expected. Harding reported that he has used the isethionate instead of the hydrochloride of pentamidine in 150 mg. doses intramuscularly in 200 cases and he has found that the unpleasant side effects of the hydrochloride are eliminated.

Delayed toxic results have been reported by several authors. Wein, Frieman and Scotcher report cloudy swelling of the kidneys and fatty degeneration of the liver following the administration of repeated high doses to animals. Daubney and Hudson produced severe fatal fatty degeneration of the liver associated with edema of the gastric mucosa in a healthy calf which received three injections of 5 mg. per kilogram of stilbamidine at seven day intervals. The calf was killed "in extremis" on the twenty-eight day. Napier and Guptal[140] reported that three to four months after the administration of stilbamidine for Kala-azar six patients developed anesthesia to light touch over the trigeminal area and one over the distribution of the third cervical nerve. This lesion was non-progressive with a tendency to spontaneous cure after several months. McComas and Martin have reported a fatal case following the administration of three injections of 100 mg. each of freshly prepared pentamidine. These were given over a period of four days. After the first two injections the patient, a 28 year old Gold Coast soldier, complained of pain in the left temple. Ten minutes after the third injection he passed into a status epilepticus. The fits were controlled with intravenous pentothal but except for two brief occasions he remained unconscious until his death 14 days later. Autopsy revealed the typical findings of trypanosomiasis (lymphadenopathy, perivascular cuffing, flattening of the brain cortex and thickening of the meninges), early fatty degeneration of the liver and bronchopneumonia. Since the patient's blood had been repeatedly negative for trypanosomes upon animal inoculation after therapy was begun the authors concluded that the death was probably due to a Herxheimer reaction.

One marked advantage of the diamidines which remains to be studied more carefully is the apparent reluctance of trypanosomes to become resistant to them. Fulton, Yorke and Lourie were able to prepare resistant strains with considerable difficulty using synthalin and undecaine diamidine and these strains lost their resistance within three years. Similar resistant strains prepared using tryparsamide and Bayer 205 have retained this characteristic for many years. These authors also demonstrated that resistance to the diamidines was unrelated to resistance to other trypanocidal drugs.

MISCELLANEOUS MEANS OF THERAPY

Neocryl: This drug (sodium succinanilomethyl-amide-p-arsonate) has been studied by Acres, Murgatroyd and MacQueen. Murgatroyd treated 122 cases, 44 of whom had normal spinal fluids. He employed a routine course of 10 doses of 0.045 gm. per kilogram of body weight given at weekly intervals. Of thirty early persons who completed the course all were clinically improved but one relapsed 16 weeks later. Forty-six late cases completed treatment and 35 of these were clinically improved. However in four cases the trypanosomes never disappeared from the spinal fluid. Visual disturbances occurred in several cases. Murgatroyd believes that the results with this drug are similar to those with tryparsamide.

MacQueen noted rapid clinical improvement in 60 cases. The blood and lymph were cleared of trypanosomes at the second examination. In two cases there were visual disturbances, one of which became entirely blind and expired within a month of cerebral trypanosomiasis.

Acres treated nine early cases with three relapses and twelve late cases with nine relapses. The period of observation lasted two years. The author states that the relapse rate in the advanced cases compared very unfavorably with that obtained using tryparsamide in the same area. Four of the relapses were retreated with no improvement and then two of them were resistant to tryparsamide.

Arsenobenzene: Arsenobenzol has been found to sterilize rapidly the peripheral blood of mice infected with *T. brucei.* Whether it is rapidly changed *in vivo* into an arsenoxide is unknown. Radna tried injecting 1 mg. of this compound into the spinal canal of seven persons suffering from sleeping sickness. This apparently increases the permeability of the blood-cerebrospinal fluid barrier thus greatly improving the effectiveness of such trypanocidal agents as tristibene and Bayer 205.

Azoarsenobenzol: Friedham treated 41 patients with eight to twelve doses of 0.5 gm. of azoarsenobenzol 4197 given every two to four days. Peripheral sterilization was complete in 38 cases after the first injection and in the remaining three after the second injection. There was rapid clinical and spinal fluid improvement and no toxic results were noted.[147] A longer period of observation will be necessary before the drug can be properly evaluated.

Triazine Arsenic Acid: Friedham and Muraz have reported very favorably on the use of the disodium salt of 2:4-diamino-6 (arsonoanilino) triazine. They used six to ten injections of 0.03 to 0.5 gm. per kilogram given every three days. Blood sterilization was rapid following one injection. Friedham treated 89 cases and obtained a rapid reduction in cerebrospinal fluid cells and a slowed drop in the albumen; 29 of these cases examined two and a half to nine months later showed no relapses. He also states that when compared with tryparsamide in mice triazine arsenic acid is eight times more active and less toxic.

Gamma-(p-arsenosopheny)-butyric acid: Eagle reports that this drug is stable in solution and in mice and rabbits has a slightly greater chemotherapeutic index than tryparsamide. In 2 per cent solution it is practically non-toxic. He has treated 200 patients in the Gold Coast, Nigeria, Belgian Congo and West Africa with six to twenty-four injections one to seven times weekly of 0.25 to 0.5 mg. per kilogram intravenously and intramuscularly. One important feature is that this compound is effective against "arseno-resistant" trypanosomes *in vitro*, in animals, and in man. Peripheral sterilization requires less than 60 minutes. The results have been gratifying in early cases but not too good in late cases where it may cause encephalitis.

Etharsanol, Proparsanol: Etharsanol (monosodium salt of 2-p-arsono-arsilino-ethanol) and proparsanol (monosodium salt of 3-p-arsono-anilino-propanol) each containing 20 per cent arsenic have been recommended for the treatment of trypanosomiasis. Stratman-Thomas, Lovenhart, and Van den Branden found etharsanol comparable in effect to tryparsamide but both of these drugs are more likely to produce ocular disturbances than is tryparsamide.

Phenanthridines: A number of compounds of the phenanthridine series have been found to be effective against *T. congolense.* Notable among these is 7-amino-9-(p-amino-phenyl)-10-methyl phenanthridinium chloride. Their effect against *T. gambiense* seems to be much less than that against *T. congolense.*

Antimonials: Many antimony compounds have been tried in trypanosomiasis. In *T. gambiense* infections these have all failed to prove as effective as the arsenicals. Among those which have been used are: stilbamine, stilbenyl, neostibosan, neostam, tartar emetic, antimony sodium thioglycollate, anthiomaline, fuadin and antimosan.

Bismuth Compounds: Kolmer, Kast and Rule have shown that the relatively high concentration of 1:2400 is required *in vitro* for immobilization of *T. equiperdum* by potassium bismuth tartrate, sodium bismuth tartrate or bismosol. The immobilization correlates fairly well with trypanocidal action as determined by mouse injection tests.

Atabrine: Atabrine (erion, quinacrin, mepacrine, chinacrin) is absorbed by trypanosomes *in vitro* and Hawking has shown that it is slightly trypanocidal *in vitro*, although Fischly and Singer state that there is no trypanocidal activity *in vitro*. De Borchgrave and others have shown that this drug and argoflavine both have a trypanocidal action *in vitro* in dilutions as high as 1:30,000 and 1:15,000. The presence of the orthoquinoid radical prompted Lourie and Collier to investigate whether there is any risk of producing resistance to the arsenic compounds and non-metallic dyes containing similar groups. They concluded that there was no such danger.

Sulfonamides: P-aminophenyl-sulfonamide, sulfanilamide, and the other sulfonamides exert no deleterious effect on *T. brucei* or *T. equiperdum*.

Penicillin: Attempts at the treatment of Rhodesian trypanosomiasis and experiments using *T. cruzi* and *T. congolense* have shown that penicillin is ineffective against trypanosomes.

X-rays: The direct lethal dose of x-rays is not the same for all types of trypanosomes. The M. L. D. varies from 100,000 r. to 600,000 r. Only 2 per cent of this amount is required to prevent normal division.

Anticoman, Surfen, Congasin-Bayer: These three compounds were reported by Brauss in 1944 following experiments begun in 1939 and interrupted by the war.

Anticoman, a guanidine derivative, failed to cure *T. brucei* infection in mice although it cleared the peripheral blood in 24 hours.

Surfen appeared to act in a manner similar to Bayer 205 although not so effectively. It also has a less prolonged effect. The author concluded that it would probably be a useful trypanocide. It was also effective against *T. equiperdum* but ineffective against *T. congolense*.

Ethylenediamines: Funke, Bovet and Montezin prepared and tested 39 compounds of ethylenediamine on mice infected with *T. brucei*. Eight of these showed some effect; the best was (p-isopropyl-benzylamino)-1-amino-2-ethane. Activity seemed to depend on the presence of the diamine group and on a second substituted group in the benzene ring.

SUMMARY

1. A brief introduction and historical sketch of the discovery of the cause of African sleeping sickness is given. Early methods of therapy, notably with atoxyl, are mentioned.

2. The early reports on the efficacy of tryparsamide are given and the best modern methods of treatment by the use of the drug are outlined. The cure rates in early and late stages and the toxic manifestations are mentioned. Combined treatment as advocated by numerous authors consists of one to four doses of 1 gm. of Bayer 205 followed after several weeks by three or more doses of tryparsamide at weekly intervals.

3. The method of action, prophylactic effect, toxic reactions and efficacy of Bayer 205 are discussed. This drug when used alone is recommended only in early cases.

4. The development, metabolic and toxic effects and clinical results of the diamidines are described. Eight to twevle doses of 50 to 100 mg. of pentamidine produces excellent results in early cases. A prolonged prophylactic action has been demonstrated.

5. A number of newer drugs are mentioned. Among those which show promise of value are neocryl, azoarsenobenzol, triazine arsenic acid, gamma-(p-arsenosophenyl)-butyric acid, etharsanol and surfen.

It has been necessary to delete the references to this article because of their large number, namely 171. The reference list may be reviewed in the office of the editor by anyone who is interested in going over this complete bibliography.

BIOTIN

BRUCE W. ALSPACH, M. D.
NEW ORLEANS

The term vitamin was first used by Funk in 1911 to describe those indispensable food substances which man cannot synthesize in adequate amounts and which, therefore, must be obtained from food. The number of these substances is large and constantly increasing so that the general practitioner and medical student has long since fallen behind in the evaluation and utilization of those vitamins which have been isolated and described recently. A paper such as this therefore serves a definite purpose in correlating the available information and appraising the value of one of the more recent vitamins—biotin.

HISTORICAL

The history of biotin goes back to 1901 when Wildiers, working in Ides' laboratory in Louvaine, Belgium, described a compound called "bios" which was essential for the growth of yeast. This compound has since proved to be multiple in nature and from it, thiamine, pantothenic acid, pyridoxine and several other substances have been fractionated.[15]

One of these fractions, tentatively labelled Bios IIb was investigated further by Kögl. He succeeded in isolating from egg yolk a crystalline methyl ester of a compound which possessed the greater part of the yeast stimulating factor of Bios IIb and which he called biotin. Kögl[45] worked extensively with the substance, describing some of its chemical and physical characteristics and gave it the empirical formula of $C_{11} H_{18} O_3 N_2 S$.

Previous to these experiments by Kögl other investigators, particularly Allison, Hoover and Burk, had described concentrates which were growth and respiration promoting on certain strains of Rhizobium, a legume nodule organism.[1] These concentrates were derived from either commercial sucrose, hydrolyzed yeast or cultures of azobacter. Since the active substance was a co-enzyme of respiration it was called "co-enzyme R." Six years later, in 1939, West and Wilson postulated that co-enzyme R and biotin were identical.[95] This fact was proved that same year by workers in Sweden.[63]

In another field of biologic investigation, apparently unrelated to the two already mentioned, Boas in 1927 reported a syndrome which occurred in rats when dried egg white was added to their diet.[3] On this regime the rats lost their hair and a dermatitis, skin hemorrhages and loss of weight occurred, followed by a marked spasticity and then death. Boas describes a "protective factor X" present in raw potatoes, yeast and liver which would both cure and prevent the syndrome. The etiology of this "egg white injury syndrome" and the nature of the protective substance remained unknown for some time, although much information was obtained by numerous groups working on the problem. Findlay and Stern[21] suggested that it was a lack of a vitamin-like substance which caused the syndrome, and Sollman and Goodman later showed that drying the egg white had no effect on the occurrence of the egg white injury since it could be produced by using the fresh material as well.[85]

The protective factor was intensively studied by Parsons and her associates[68] who reported that the toxic factor in egg white was apparently protein in nature. They elaborated on the symptomatology in the rat,[49] and other animals,[50] and described further the protective factor.[69, 70] György and co-workers reported on the chemical and physical properties of the protective factor which he called "vitamin H", from the German "haut" meaning skin, referring to the dermatitis produced in the deficiency state.

Early in 1940, György joined the Cornell group of du Vigneaud and Melville. A short time later, the possible identity of vitamin H with biotin and co-enzyme R was suggested and in that same year the fact was established by vitamin H assays of a sample of biotin supplied by Kögl.[33] This was confirmed by the demonstration of the vitamin H activity of biotin from liver concentrates which contained a high index of vitamin H.[29] Thus three ostensibly different fields of investigation each concerned with an extremely biologically active compound with well-defined actions were brought together by the demonstration, through persistent and often brilliant research, of a common identity and common actions.

CHEMISTRY

In spite of the progress made in identifying and analyzing biotin, only the empirical formula suggested by Kögl in 1938 was available. This formula was subsequently modified in 1942 by du Vigneaud[16] and associates to $C_{10} H_{16} N_2 S$.

This compound, which is a monocarboxylic acid of high molecular weight, appears in crystalline form in long, thin, plate-like needles. It has a melting point of 230°—232° and is bicyclic in structure, one ring representing a five membered urea structure and a ring sulphur in a thio ether linkage possessing a six carbon straight moiety. Biotin is optically active and is easily destroyed by hydrogen peroxide, by peroxide containing ether and by nitrous acid.[28] Rancidified fats will also inactivate pure biotin, apparently by a partial oxidation. The end product is the same as that obtained when hydrogen peroxide is allowed to act on biotin.[73]

Pure biotin is one of the most active substances known, its effect on certain yeast strains being noticeable in a concentration of one in four hundred billion. This may be contrasted with the effect of ephinephrine on the frog's pupil in a one in twenty million dilution.[40]

As would be expected from the amount of work done on biotin, a large number of similar and related substances have been produced. The monomethyl ester of biotin was prepared from liver by du Vigneaud and his associates in 1941.[17] This is similar to the crystalline derivative of Kögl[46] prepared from egg white. The difficulty of obtaining biotin for investigation is exemplified by the fact that only one milligram was produced from a quarter of a ton of dried egg yolks.

In addition to the methyl ester, desthiobiotin has been isolated, a compound similar to biotin with the sulfur atom removed and replaced by two hydrogen atoms.[54] It has approximately the same activity as biotin on certain micro-organisms, but does not stimulate the growth of *L. casei* unless biotin is present. A large quantity of desthiobiotin, however, will inhibit the action of biotin.[51, 55] This would suggest that certain organisms have the ability to convert desthiobiotin to biotin and utilize this latter substance. Leonian and Lilly have shown that where desthiobiotin is capable of replacing biotin, the micro-organisms utilize only 40 per cent of the available substance present.

Less important related substances, some of which are isomers of biotin, are dl-biotin which is half as active as biotin, 1-biotin which is one-fourteenth as active and allobiotin which is inactive. O-heterobiotin is an analogue of biotin in which the S in the thiophene ring is substituted by an O. It is about one-fourth as active as biotin.[14] Biotin sulfone has about 0.1 per cent of the growth promoting properties of biotin and must be given in tremendous quantities to cure egg white injury.[12] Diaminocarboxylic acid is derived from biotin by heating it with an alkali under proper conditions, thus splitting the urea ring. It has from 3.6-10 per cent of the activity of biotin.[19]

After the identification of the specific molecular structure of biotin it was only a matter of time before this substance could be synthesized. Success in this was first reported by Harris, Wolf, Mozingo and Folkes[34] in 1943. Subsequently investigation has proved this substance to be identical in every respect with the natural occurring material.[86, 66, 19] The fact that the synthetic substance has in its make-up three different asymmetrical carbon atoms makes possible the existence of several stereochemical isomers related to biotin, all with varying activity, of which a few may be listed. Synthetic dl-desthiobiotin is 50-60 per cent as active as the natural compounds but has only 0.1-0.01 per cent of the activity of biotin in rats.[80, 101] The synthetic equivalent of diaminocarboxylic acid, dl-diamino acid sulfate, is 4-7 per cent as active as biotin.[86]

METHODS OF ASSAY

The earliest methods of assay were both cumbersome and time consuming, for although biotin occurs widely in nature it is in exceedingly small concentrations. Animal assays, using the rat, were utilized by György[27] for his determinations of vitamin H. The chick has been suggested for the same purpose,[29] but these methods are no longer used and those based on the acceleration of growth of micro-organisms by biotin are in vogue at present. There are no chemical methods available for this determination.

Kögl measured the growth of standardized yeast strains over a five hour period as an assay method. In 1940, Snell, Eakin and Williams[84] described the most popular procedure in use today. They utilize the measured growth of a small seeding of Saccharomyces cerevisiae after a twenty hour incubation period in a synthetic media devoid of biotin. The incubation period has since been reduced to sixteen hours and Hertz[37] has widened its range of usefullness. The use of micro-organisms for the assay of biotin has been suggested and includes *Lactobacillus arabinosis*,[103] *Rhizobium trifolii*,[96] *Clostridia butylicum*[46] and *Lactobacillus casei*.[83] This last named organism has been used by Peterson and associates as a basis of a micro-biological test which they report to be accurate to within ten per cent. It has been shown since to have a wide application for assays of vitamins in the B complex group.[47]

In these micro-biological tests biotin should be present in the free form and not as the methyl ester which slows the growth and fermentation of the yeast.[87] A pH of 6.8 has been found most favorable for the estimation.[29] Extraneous growth stimulants which might cause an error in the determination are thought to be lipoid in nature and may be removed by simple filtration of the aqueous extracts.[98] Trager has shown that these methods are not applicable to the estimation of biotin in the blood.[93]

DISTRIBUTION

As has been previously stated biotin occurs extensively in nature, always however, in low concentrations. This is shown by the fact that liver, the richest source of biotin, contains less than 0.0001 per cent of the vitamin. The only other foodstuff comparable to liver in its content of biotin is the kidney. Beef, cereals, yeast, peptone, egg yolk, malt and alfalfa, all have comparatively large amounts of biotin. Chicken meat, vegetables and fruits, however, offer only fair quantities. Cooking appears to affect the biotin content to a minimal degree. Schweigert and co-workers found that 77 per cent of the biotin content of meat was retained after cooking. From 1-5 per cent of the biotin was recoverable from the drippings, making a total retention of 78-82 per cent of the original biotin content of the meat.[81]

Separate sections will be devoted to the occurrence of biotin in the bowel and tumor tissue, urease, muscle phosphorylase or any of the crystalline enzymes-catalase, renin, urease, muscle phosphorylase or concanavillin A.[56]

BIOLOGICAL SIGNIFICANCE IN YEASTS, VACCINIA AND BACTERIA

Biotin is a respiratory catalyst and as such is an essential nutrilite which most strains can synthesize to some degree.[44] The extent of this ability, however, is dependent on the synthesizing abilities of the cells used and the composition of the medium. It is reasonable to believe that, since all the synthetic processes of a cell are closely related, the presence or absence of one essential metabolite would affect the nutritional requisites of the others. Williams, Eakins and Snell have shown that biotin synthesis was facilitated by the presence of other essential metabolites such as, inositol, thiamine, pyridoxine and pantothenic acid. While synthesis did occur before the addition of any of these metabolites it was at a very slow rate.[99] To repeat, therefore, it cannot be said that the nutritional need by yeasts for any one substance of this group is absolute, but rather dependent on the presence of other substances. The ability of certain strains of yeast to convert desthiobiotin into biotin has already been stated.

A great deal of work remains to be done in order to clarify the importance of biotin to vacinnia. Hoagland and associates[39] have shown that elementary bodies of vacinnia possess a concentration of biotin approaching that of egg yolk, liver and molasses. They foud a marked concentration of biotin parallel with an increase in viral activity. We know therefore, that biotin is intimately associated with the enzyme systems of vacinnia, although how essential it is has not yet been determined.

Bacteria may be compared to the yeasts in that biotin is an essential substance required by them. Their need for an exogenous supply of this vitamin is governed almost entirely by the relative ease with which they are able to synthesize it. In a strict sense, it would be incorrect to separate the bacteria for which biotin is essential, as determined by a complete absence of growth in a biotin-free medium, and those bacteria for which biotin merely acts as a growth stimulant. This latter phenomenon is due to the slow rate of synthesis by the bacteria, allowing a stimulation to be seen on the addition of biotin.

Those that cannot synthesize biotin and require an exogenous supply are certain strains of Rhizobium, Clostridium, Lactobacillus, Streptococcus and Brucella. Certain strains of Proteus and *Staphylococcus aureus* are capable of synthesizing biotin slowly and therefore they show only a slight stimulation on the addition of biotin. *Mycob. tuberculosis, B. subtelis, E. typhosa, Esch. coli, B. anthracis* and strains of Pseudomonas all synthesize biotin easily and show no change when biotin is added to media containing them.[44] In spite of these widely varying powers of synthesis, Thompson[91] has shown that the bacteria cell content of biotin remains constant. We may assume then, that the production of biotin by bacteria, which Thompson has shown to parallel the rate of bacteria growth, is a result of overproduction and not cell autolysis.

A large percentage of the bacteria capable of synthesizing biotin are normal inhabitants of the intestine of both man and animals. Wegner and associates[94] have shown that biotin is synthesized in large amounts in the rumen of the calf. The biotin content of the rumen is about five times greater than the original a few hours after feeding. The rumen of the cow and the cecum of the rat are the main centers of biotin synthesis in these animals.[53, 58] In man this synthesis takes place in the terminal ileum and large gut.

The efficiency of this synthesis is very high since rats on a biotin-free diet thrive well. Those on a biotin-low diet are able to excrete the vitamin in amounts that greatly exceed the intake.[60] Similar results in man have been described by Opell[64] who reported that three to six times the biotin content of the diet was excreted in the urine and stools of his subjects.[64] The work of Najjar and Holt[59] suggests that the bacterial synthesis of biotin in man is adequate for his needs.

The diet will obviously affect the quantity of synthesis taking place in the bowel both by changing the media upon which the bacteria act and by causing marked differernces in the intestinal flora itself, with corresponding changes in the products of those bacteria. Dextrin and dextrin containing carbohydrates appear to promote intestinal synthesis, as does roughage which increases the amount of unabsorbed food substances that provide an ideal medium.[58, 26] The interrelationship between the various vitamins is demonstrated in the rat by the decrease in biotin synthesis when a pantothenic acid or riboflavin-low ration is used.[61]

AVIDIN

Boas used egg white to produce the first biotin deficiency syndrome in rats. It has since been shown that this is due to the formation, in the gastrointestinal tract, of a non-absorbable complex between biotin and a constituent of the egg white. This substance which was named 'avidin' because of its avidity for biotin has been isolated in crystalline form. It is a basic protein of high molecular weight which combines with the biotin in a ratio of one molecule of biotin for one molecule of avidin.[102] The avidin-biotin complex (AB) cannot be dissociated in the digestive tract since it is unaffected by the proteolytic enzymes pepsin, trypsin, pancreatin and papain.[30] It is also unaffected by changes of temperature and acidity and is therefore excreted in the stool.[31]

The only way that biotin may be released from this combination is by steam sterilization or by irradiation in the presence of riboflavin.[32, 48] In vivo this may also be accomplished by parenteral administration of the complex, apparently thus making biotin available to the body since egg white injury may be cured in this manner.[32] Proteins associated with eggs are the only ones which exhibit this pronounced biotin combining tendency.[38] It is interesting to note that avidin will inhibit the growth of those bacteria for which biotin is essental and affect only minimally those that are able to synthesize satisfactory amounts of the vitamin.[48]

The nature of the biotin-avidin combination is still unknown. It has been suggested, however, that both the cyclic urea and the aliphatic side chain are essential for the formation of this complex. When they are hydrolyzed, as in diaminocarboxylic acid, a combination between this acid and avidin will not take place.[13]

The question of the existence of an avidin-uncombinable biotin is still open to speculation. There is some evidence that large amounts of rat or human urine contain quantities of such material but it has not yet been proved that it has biotin activity or that, since it is found only in the urine it is not a partial break-down product of biotin.[8]

IN RATS

Induced biotin deficiency in rats is essentially the egg white injury syndrome which has been produced and described many times.[20, 62, 71] Characteristically, within the first two or three weeks on a synthetic diet, which ordinarily allows good growth, to which ten per cent egg white has been added, there occurs a loss of the long guard hairs of the body, leaving a soft mole-like coat. This is usually accompanied by an exfoliative, pruritic dermatitis, commencing on the abdomen and progressing to a generalized alopecia. A change in the color of the fur may also take place. Frequently, red scaly patches form at the corners of the mouth together with a reddening and swelling of the eyelids, giving a "spectacle eye" appearance. The animal's weight begins to fall and symptoms referable to the nervous system appear in the second or third week of the deficiency. The back becomes arched and the limbs spastic giving an abnormal "kangaroo-like" posture. Terminally there is a rapid loss of weight and cyanosis. Histopathologically in the acute deficiency state there is early atrophy of the thymus and testes as a manifestation of an ageing process. The skin shows marked drying and scaling with almost complete sloughing of the stratum corneum. Liver stores of biotin are greatly reduced and the stores of body fat are almost completely depleted.[3] Skeletal muscles are pale and striated and atrophied terminally.[82] The creatine content of the muscles has been shown by Pizzolato and Beard[76] to decrease on an average of 20 per cent in 136 days of a biotin deficient diet.

Despite the extreme spasticity observed clinically no degeneration of the spinal cord or peripheral nerves exists, nor is there at any time during the deficiency an impairment of the motor nerve to elicit tension in its muscles. It may be noted at this time that all these changes are reversible and the administration of crystalline biotin quickly brings about a complete remission of symptoms.

Daft, Ashburn and Sebrell[10] first noted that the symptoms of biotin deficiency could also be produced in rats by the administration of sulfaguanidine or sulfasuxidine in purified diets. This deficiency is usually complicated by deficiencies in other vitamins, particularly in folic acid, which are synthesized in the intestinal tract. Frequently, too, leukopenia and kidney lesions develop as specific effects of the sulfonamides and these may be relieved, not by biotin but with other measures, including the administration of whole liver. Remission of this deficiency or a protection from it could be obtained by treatment with biotin and sometimes folic acid.[57] These findings have been confirmed by other workers.[52]

The mechanism of the production of this deficiency is predictable when the role of intestinal synthesis of the vitamin is recalled. The sulfonamides used are non-absorbable and as such act chiefly on the bacerial flora in the intestines, reducing their number and thereby reducing the amount of vitamins synthesized. This leads to the production of a deficiency. The concept has received almost unquestionable support and has a large amount of experimental evidence to corroborate it.[11] There is no proof, however, that the inhibition of the bacterial synthesis of biotin and not some other factor of the sulfonamides caused the deficiencies. It may also be noted that there are other observations which are difficult to reconcile with this theory.[10]

The mode of action of biotin in animal tissues appears to be as a component of an enzyme system concerned with the metabolism of pyruvate. On a biotin deficient diet rats show decreased rates of oxidation of pyruvate by the liver.[75] This is similar to what occurs on a pantothenic acid deficient diet and therefore suggests a close relationship between these two vitamins. Wright and Welch[104] have shown that pantothenic acid cannot be utilized in the rat except in the presence of biotin.

There is also evidence that biotin is concerned with fat metabolism. Gavin and McHenry[25] have obtained fatty infiltration of the liver of rats by the administration of biotin. The fatty livers were characterized by a high cholesterol content. The authors concluded that biotin does not augment the quantity of cholestoral synthesis but does increase its concentration in the liver.

IN AVES

In contrast to animals, birds are not capable of synthesizing biotin in their intestines. The deficiency syndrome may be produced by a nutritional absence alone as well as by the administration of avidin contained in egg white.[2, 35, 72] The deficiency syndrome consists almost entirely of skin changes. There is bleeding and cracking of the skin of the feet, reddening and swelling of the eyelids, loss of feathers and the appearance of a bloody scab on the mandible.[49] Cravens and his associates[9] have shown that biotin-deficient chickens produce eggs with a high proportion of chondrodystrophy, syndactyly, crooked tibia and twisted tarsus. Embryonic mortality was also higher, the peaks occurring on the third day and the last three days of incubation. Usually this picture is complicated by a perosis which many authors do not believe is due entirely to biotin deficiency, and which may be prevented by the use of small amounts of choline.[79, 36] The dermatitis may be relieved by treatment with biotin or its methyl ester.

Trager has shown that biotin deficient chicks and ducks are more susceptible to infection with Plasmodium lophurae, have higher "peak parasite numbers" and greater mortality than the controls. Older chicks, which have a higher biotin content of their tissues are less susceptible to *P. lophurae* infection than the younger ones. The biotin content of the blood rises after infection, possibly indicating the elimination of the parasite from the blood. The mechanism for this is, as yet, unknown.[93] Trager's work has been confirmed and György has reported essentially the same results with *T. lewisi* in the rat.[41]

IN HUMANS

The importance of biotin in human nutrition has received considerable attention. Findlay and Stern[22] first suggested that biotin was in some way associated with Swift's disease in children. Appel has reported that there is little difference in the total urinary biotin of normal individuals and those with a variety of diseases. The normal range for biotin in the urine in 24 hours is 14-111 mg. and in the feces for the same period, 86-191 mg. These figures are about three to six times the intake from the diet, thus indicating the synthesis of biotin.[65]

Keller[41] has noted a relationship between biotin and skin sensitivity, and Williams[10] reported a clinical case of biotin deficiency in a patient with dermatitis exfoliativa.

This patient was on a restricted diet, a large proportion of which consisted of raw eggs. Rhoads and Abels[78] failed to induce a biotin deficiency or even reduction of urinary biotin in two patients who were fed for thirty weeks sixteen to forty times the amounts of avidin necessary to bind *in vitro* the limited amounts of biotin in their diets. Peterson and his associates[74] have shown that there is always an excess of output of biotin over intake, the degree being dependent largely on the diet. The greatest excess of output over intake occurs on a low biotin intake and the least excess occurs on a high biotin intake during liver feeding.

The most definitive work on this subject has been done by Sydenstricker and co-workers.[88, 89, 90] They maintained four humans on a low vitamin diet of which 30 per cent of the total caloric intake was supplied by egg white. On this diet the subjects developed a desquamative dermatitis and pronounced pallor of the skin and mucous membranes. These symptoms appeared in approximately ten weeks. The biotin secretion in the urine diminished to almost a tenth of its normal quantity. The blood showed a diminution in hemoglobin content, red blood cells and hematocrit, in spite of theoretically adequate amounts of iron and protein in the diet. The pathological conditions were rapidly cured by the administration of 150-300 micrograms of biotin daily. Unfortunately, definite conclusions cannot be drawn from this work since the biotin used was not pure.

IN TUMORS

For many years the effects of various vitamins which are capable of exerting a powerful regulatory influence on respiration and growth have been studied in relation to tumor metabolism. The first link between biotin and cancer was described by du Vigneaud and associates. It was shown that biotin had a procarcinogenic effect on hepatomas produced in rats with the dye dimethylaminoazobenzene, more commonly known as "butter yellow."[18] Ovalle[97] substantiated this by showing that on the addition of biotin to the diet of rats receiving butter yellow and protective vitamins, half of them developed tumors. This was surprising since Kensler and co-workers[77] previously found that the inclusion of dried liver or yeast in the diet of the rats would completely nullify the effects of the dye when it was used.

In later studies du Vigneaud showed that avidin in egg white had a strong anti-carcinogenic effect. One explanation is that the egg white does not owe its protective effect solely to the avidin but is itself capable of partially detoxifying the dye.[43] Other workers[97] have since reported the biotin content of tumors to be higher than that of normal tissues. Embryonic tissue, which, in common with tumors, possesses the ability to grow rapidly, also contains relatively large quantities of biotin. West and Woglom[96] have concluded that the biotin level of tumors deviates from the normal adult values in the same direction as that of corresponding embryo tissues.

The attractive hypothesis, however, that by a reduction in the biotin supply an inhibition of tumor growth may be accomplished has not proved to be entirely true. In the work of Rhoads and Abel[78] already cited, one patient had a chronic lymphocytic leukemia and the other an inoperable cancer of the breast. No clinically evident biotin deficiency was secured and the course of the neoplasms was not altered. The patient reported by Williams[100] as having the only naturally occurring biotin deficiency on record, subsequently developed a carcinoma of the penis. By maintaining tumor-bearing mice on egg white diets West and Woglom found that the biotin content of the tumor could be markedly reduced with no great influence on the growth of the tumors.

Doubt is expressed by du Vigneaud that tumor tissue ever has a higher biotin content than normal tissues.[43] Pollock and his associates [77] determined the biotin content of a great many neoplastic and normal tissues and concluded that in all probability, cancers neither require nor are associated with a high biotin content.

More recently, an explanation has been offered for the obvious discrepancy which exists in the biotin content of tumors and normal tissue. Burk and Wingler[4] have shown that there are substances occurring naturally in normal tissues which are chemically similar to biotin. These substances do not cure biotin deficiency in animals or combine with avidin. Thus it is possible that reports showing a higher biotin content for normal tissues may be the result of inadvertently measuring both true biotin and the animal-inactive, avidin-uncombinable biotin forms. These authors[5] state that the percentage of avidin-uncombinable biotin in normal and tumor tissues of biotin deficient rats rises to many times the normal values of 1 to 5 per cent, up to as high as 80 to 90 per cent.

In passing, it is of interest to note that biotin, in the living animal or in tissue cultures, will not affect the rate of axon growth.[6, 23]

CONCLUSION

In attempting to appraise the value of biotin to humans, one must primarily remember that a great number of investigations on this subject are still in progress, the results of which may radically change our concept of this vitamin. At present, however, one fact stands out as of paramount importance, namely, that the intestinal synthesis of biotin appears to be adequate for human needs. A natural deficiency can evidently occur only under bizarre circumstances and even an induced deficiency is difficult to produce. Therefore biotin seems to be of importance in a secondary nature only, that is in its relation to tumors. No clinical deficiency has as yet been produced in a human with a neoplasm and it would therefore be unjustifiable to attempt to draw a conclusion at the present time. It is doubtful that sub-clinical deficiency states of this vitamin exist to the degree of making disease processes, where they exist in conjunction with this state, develop into a more fulminating nature. The future of biotin appears to lie chiefly in its clinical effect on neoplasms and, what is perhaps more important, in the further investigation of related chemical substances. Of these, avidin in particular bears watching and with time it may well attain a position out of all proportion to the one it now occupies.

SUMMARY

1. Biotin is a recently isolated member of the vitamin B complex.

2. It is essential to the life and growth of some bacteria and yeasts, apparently being a co-enzyme of the respiratory metabolism.

3. The specific chemical formula and molecular structure have been determined.

4. Yeast growth assay methods are the best in use at the present time.

5. Biotin is widely distributed in nature although in minute quantities.

6. Bacteria have varying abilities of synthesizing biotin.

7. Biotin deficiency causes a specific syndrome in various animals and man.

8. Its activity is opposed by avidin, a protein constituent of egg white.

9. Bacterial synthesis of biotin in the intestinal tract is adequate for human needs.

10. The deficiency state in *Aves* increases the susceptibility to and course of malaria.

11. It may be a procarcinogenic agent.

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HEALTH AND MEDICAL LEGISLATION

There has been sent out by Marjorie Shearon, Ph. D., who signs herself as Research Analyst, Conference of the Majority of the United States Senate and gives her address as Senate Office Bldg., Room 8B, two mimeographed discussions of current developments in the United States Senate. We do not know who Miss Shearon represents but we do know the material that has so far appeared is attractively prepared, most readable and decidedly in favor of the medical profession. In bulletin No. 2 Miss Shearon points out that the revised Taft-Ball-Smith Bill, the National Health Act of 1947, is now ready for introduction. This calls for the establishment of a National Health Agency which would be under the Department of Health, Welfare and Education which will have three under secretaries and eventually a Cabinet officer who will be in charge of the welfare department. She speaks of this as nothing more or less than a glorified Federal Security Agency which would be the worst possible set-up for health and medical activities. If such Cabinet position is established it seems almost sure that it will be given to a welfare official, "to a social insurance devotee, or to a welfare official, combining the social worker's viewpoint with a bias in favor of national social insurance." Miss Shearon mentions the fact that Watson B. Miller, the present Federal Security Agency Administrator, is a strong proponent of compulsory sickness insurance such as the Wagner-Murray-Dingell Bill advocates and he is a very likely candidate for the Cabinet post.

If it should come about that the welfare department should be established as a Cabinet position it would look as if this would be the end for the time being at least of a much to be hoped for Cabinet set up in which a physician would represent the various organizations which have to do with the care of the sick. This is a measure which intelligent physicians have been advocating for years but it would all go by the board if a welfare department should be established. It would not only see the passing of any immediate representation of the medical profession in the Cabinet but actually it would mean that the medical profession would probably be worse off than it is at the present time in so far as bureaucratic control of medical activities is concerned. There should be a separate health agency. Undoubtedly the American Medical Association would be in a position to combat the ideas that are represented in the Taft-Ball-Smith Bill. Nevertheless it would be wise to accept Miss Shearon's ideas for members of the medical profession to express their views as individuals to their Representatives and Senators.

BAL

The medical profession is well acquainted with the advances that have been made in therapeusis in the past few years, notably in the management and treatment of infectious diseases. Very extensive researches are now under way or about to get under way to study the effect of antibiotics in the treatment of tuberculosis. It is rather unusual, however, to find that certain new and almost unheard of drugs have been employed in other disorders which are almost as magical in their effect as are the sulfa preparations, penicillin and streptomycin in the infectious diseases.

BAL is one of the new drugs that because of war secrecy has only recently been announced to the medical profession. BAL is an abbreviation for British Anti-Lewisite, a preparation which was developed as an antidote to arsenic blister gases. It was intended entirely for local decontamination but was found later to be extremely effective in the systemic treatment of severe arsenic poisoning. Arsenic poisoning might occur after hypothetical exposure to arsenical blister gases but the drug has been found to be of tremendous value in the treatment of the complications that result from arseno-therapy. It has also been found to have remarkably powerful protective and therapeutic value in the treatment of mercury poisoning.

The literature on BAL is very scanty but so important was it considered to put before the American medical profession the clinical uses of BAL (2, 3-dimercaptopropanol) that some fifteen articles were recently published as a supplement of the Journal of Pharmacy and Experimental Therapeutics, and in the Journal of Clinical Investigation for July practically the entire issue was devoted to experimental and clinical studies of BAL by those who were given the drug for special investigations.

The clinical reports of the use of this drug are almost unbelievable. Eighty per cent of patients with typical arsenical exfoliative dermatitis showed definite improvement within three days and were fully recovered in approximately two weeks. Of fifty-five patients with hemorrhagic encephalitis as result of intensive arsenic treatment, forty of whom were apparently on the point of death, forty-four recovered within twenty-four hours to seven days. In the more severely poisoned patients BAL was administered parenterally. The compound has been used locally but for the most part intramuscular injection is the method of choice.

A group of twenty-three patients, after a study of the drug on the treatment of experimentally produced mercury poisoning in the animal, were given BAL after ingesting bichloride of mercury in doses sufficiently large to have resulted in death in the majority of them. Actually what took place was that only one patient died.

It is quite possible that BAL may be equally beneficial both in preventing and treating poisoning from other heavy metals. The drug should be made available and should be utilized by those who are treating large groups of patients or even an occasional patient who may have syphilis. Certainly it should be in the accident room of any hospital which may be called upon to treat a patient who has taken bichloride of mercury.

MORTALITY FROM DEGENERATIVE DISEASES

It is a well known fact that death rate from infectious diseases has markedly diminished, notably in pulmonary tuberculosis, and also in the last few years since the development of new chemotherapeutic agents, in pneumonia, meningitis and other coccal diseases.

Typhoid fever and most of the bacillary diseases are well under control through public health measures to ensure a pure water supply, proper disposal of excreta and vaccination of school children. Within the last ten years there has been over a 30 per cent decrease in infant and maternal mortality. The consequence is that life expectancy has definitely increased in the last few decades. Improvement in mortality has been most pronounced in the younger age groups. In 1915 the mortality rate per 100,000 in young males was 30, now it is around 10. There has been a corresponding decrease in the death of young females.

As result of the increased age incidence of the population as a whole, one would expect and anticipate that degenerative diseases would be responsible for a goodly proportion of the deaths that take place in this country nowadays. To a certain extent this is true. Heart disease leads all other diseases as a cause of death, and the heart disease that causes the death is usually that which is dependent upon ageing. The impression is quite general among physicians that diseases of the heart, arteries and kidneys now cause as many or more deaths than they did thirty years ago, but according to the statistics of the Metropolitan Life Insurance Company there has been a marked decline in mortality in these diseases in the past three decades. The death rate from this important group of causes has dropped approximately 30 per cent. The greatest reduction in mortality from cardiovascular-renal disease has been particularly notable among the white female group. The statisticians of the Metropolitan Life Insurance Company attribute the decreases in mortality, particularly in early and middle adult life, to the absence of complications as result of childhood infections. Other causes are responsible in part as well, as for example the reduction in syphilitic heart disease as result of enlightened therapy.

It should be borne in mind that while there has been an actual decrease in the mortality rate, the true death rate from cardiovascular-renal disease is higher than it was a third of a century ago but this is before statistical correction was made for the increased average age of the population as a whole.

These figures are enlightening and instructive to the physician. From the point of view of the economist and of the legislator it is obvious that old age pensions and the payment of Governmental money to the indigent must be materially increased in the future because of the increased age of the general population.

COMMENTS ON A RECENT EDITORIAL

An editorial appearing this past fall, in discussing the work of a certain state health official, was based on certain information received through the editorial office. It is to be regretted by the editor that certain facts have lately come to his attention concerning this ex-health official that were not in accord with the information that the editor had received. The recent information would indicate that the editorial of favorable comment caused considerable discussion among the members of the Medical Society because the former health official had by no means always cooperated with organized medicine. This editorial we regret very much. The editor wishes to mention definitely that the best interests of the State Society come primarily above any and all interests affecting officers of bodies not directly associated with organized medicine.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 ANNUAL MEETING

Plans are now being made for an outstanding meeting of the State Society to be held in New Orleans May 12-14. An unusual scientific program will consist of symposia conducted by three prominent out-of-state guests, Drs. Harry Gold, of New York, Maxwell Finland, of Boston and Raymond L. Gregory, of Galveston, and papers presented by members of the organization before general and specialty group meetings. Further details will be carried in subsequent issues of the Journal.

Members planning to attend the meeting should make hotel reservations, by communicating with the office of the Secretary-Treasurer, without delay as there are only a limited number of accommodations available.

REPORT OF COMMITTEE ON REVISION OF CHARTER; CONSTITUTION AND BY-LAWS OF LOUISIANA STATE MEDICAL SOCIETY

Following is revised copy of the Charter, Constitution and By-Laws prepared by the special committee appointed for this purpose. This is published in this issue in order to comply with provisions for amending the Charter, namely that a copy be sent to each member two months prior to the annual meeting at which time final action is to be taken. It is felt by the committee that it is wise to publish the Constitution and By-Laws so that the delegates may be familiar with the revision made in these also.

CHARTER

State of Louisiana,
Parish of Orleans,
City of New Orleans.

PREAMBLE

Be it known that on this tenth day of June, in the year of our Lord, nineteen hundred and three and of the Independence of the United States of America the one hundred and twenty-eighth, before me,

Carl C. Friedrichs, Notary Public, duly commissioned, sworn and qualified, in and for the Parish of Orleans, State of Louisiana, and in the presence of the witnesses hereinafter named and undersigned,

Personally came and appeared the persons whose names are hereunto subscribed, who severally declared that, availing themselves of the laws of this State upon that subject, they have formed and organized and by these presents do form themselves into and constitute a corporation for the objects and purposes, and under the stipulations and agreements hereinafter set forth and expressed, which they hereby adopt as their Charter, to wit:

ARTICLE I

Name

The name of said corporation shall be the "LOUISIANA STATE MEDICAL SOCIETY," and its domicile shall be the City of New Orleans.

ARTICLE II

Objects and Purposes

The objects and purposes of this corporation shall be to federate and bring into one compact organization the eligible members of the medical profession of the State of Louisiana, by the organization in the various districts and parishes of local branches known as component societies, which shall receive charters from this Society, but which shall not thereby be corporate bodies; and to unite with similar associations in other States to form the American Medical Association, with a view to the extension of medical knowledge, to the advancement of medical science; to the elevation of the standard of medical education, and to the enforcement of just medical laws; to the promotion of friendly intercourse among physicians, and to guarding and fostering their material interests, and to the enlightenment and direction of public opinion in regard to the great problems of State medicine; so that the profession shall become more capable and honorable within itself and more useful to the public in the prevention and cure of disease and in the prolonging and adding comfort to life.

ARTICLE III

Powers

Under the above entitled name, and for the purposes above expressed, the said corporation shall have power and authority to make, have and use a common seal, and the same to break, alter and amend at their pleasure, and by the name, style and title provided and declared, shall be capable in law to sue and be sued, and shall be empowered to make rules, by-laws and ordinances, and to alter, amend and change the same at pleasure,—but until so altered or amended, the regulations adopted at the last annual meeting shall be the regulations of the Society,—and to do everything needful for its good government and support. It shall be and is empowered to hold, lease, receive, purchase and convey all manner of property, both real and personal, and to mortgage and otherwise encumber the same; to borrow or lend money, issue bonds and notes, accept and receive donations, legacies and bequests; to own, publish, conduct and circulate such literature as will further its expressed purposes; to name and appoint such managers, servants and employees as its interests and convenience may require; to further these expressed objects and purposes by issuing charters as hereinbefore provided to such component societies as may be formed within the various districts and parishes of this State; and otherwise to do and perform all acts and things requisite and necessary to carry out the objects and purposes of said corporation.

ARTICLE IV

Membership

The active members of this Society shall be the active members of the component district and parish medical societies. In parishes where no component society exists any reputable and legally registered regular physician may become an active member provided his application has been endorsed by two active members of the Society in good standing and approved by the Executive Committee. After the organization in the parish of a component society he shall become a member of such society. The liabilities of the members shall be limited to the unpaid balance due by them to the Society. Power is hereby delegated to the House of Delegates or the General Body to create any other form of membership.

ARTICLE V

Officers

The officers of this corporation shall be a President, President-elect, three Vice-Presidents, Secretary-Treasurer and Councilors, one from each congressional district, the Chairman of the House of Delegates, the Vice-Chairman of the House of Delegates and the retiring President of the Society. These officers, and the members to be presented to the Governor for appointment on the State Board of Medical Examiners, shall be elected by the House of Delegates at the annual meeting by a majority vote of the delegates present. The President, President-elect, Vice-Presidents, Chairman of the House of Delegates and the Vice-Chairman of the House of Delegates shall be elected for a term of one year, the Secretary-Treasurer for a term of not more than five years and Councilors for terms of two years each; the Councilors being divided into two groups so that as nearly as possible one-half shall be elected annually. All of the officers shall serve until their successors are elected and installed. A person shall not be elected to any office who is not in attendance upon the annual session (except he has been a faithful attendant at the annual sessions and has a valid excuse), and who has not been a member of the Society for the past two years. Vacancies due to death, resignation, or other causes, shall be filled for the unexpired term by appointment of the Executive Committee. The Executive Committee shall also recommend names to the Governor for vacancies occurring by death, resignation or other causes on the State Board of Medical Examiners between the annual session.

ARTICLE VI

Executive Committee

The powers of this Society shall be vested in an Executive Committee, which shall be composed of the President, President-elect, three Vice-Presidents, the Secretary-Treasurer, the Councilors, the Chairman and the Vice-Chairman of the House of Delegates, who shall be ex-officio members of said Committee, and the retiring President of the Society, who shall serve as a member of said Committee for one year from the date of his retirement from office. They shall hold and exercise all powers not otherwise delegated. In cases of dire emergency when it is impossible for the House of Delegates to meet in annual or special session, the Executive Committee shall assume the duties invested in the House of Delegates by this Charter, Constitution and By-Laws. They shall meet at the domicile of the Society at least twice between annual sessions at the call of the President. Special meetings must be called by the President or the Secretary-Treasurer on the written request of four members of the Executive Committee.

ARTICLE VII

Legal Representative

The President of this Society, or, in his absence, the Vice-Presidents in order, or in their absence, the Secretary-Treasurer, is hereby designated as the officer upon whom citations may be served.

ARTICLE VIII

Life of Corporation; How to Dissolve

This corporation shall exist and continue for a period of ninety-nine years, unless sooner dissolved by a two-thirds vote of the entire membership, at a meeting specially called for that purpose after thirty days' public notice. In case of such a vote dissolving this corporation, the said meeting shall, at the same time that such vote is taken, elect three liquidators to settle and wind up its affairs.

ARTICLE IX

Amendments

The Society may amend any article of this Charter by a two-thirds vote of the active members registered at any annual session, provided that such amendment shall have been presented at a meeting of the House of Delegates at the previous annual session, and that it shall have been sent officially to each member, district and parish society, at least two months before the session at which final action is to be taken, and provided that said amendment is recorded and otherwise proceeded with as required by law.

In testimony whereof, the said parties have hereunto affixed their signatures, together with me, Notary Public, and the subscribed competent witnesses, on the day and date first above written and after reading the whole.

(Original Signed):

E. D. Friedrichs,	A. W. DeRoaldes,
John J. Archinard,	Fred J. Mayer,
P. E. Archinard,	S. L. Williams,
P. W. Parham,	J. M. Barrier,

D. L. Pothier, M. D.,
Edwin J. Graner,
S. M. D. Clark,
L. C. LeBeuf,
C. D. Simmons,
F. A. Larue,
R. Matas,
Wm. M. Perkins,
E. Denegre Martin,
J. A. Storck,
Andw. G. Friedrichs,

per pro. Wm. M. Perkins
L. B. Arceneaux,
C. M. Sitman,
J. F. Buquoi
per pro. A. G. Friedrichs,
Chas. Chassaignac,
Homer Dupuy,
Jno. B. Elliott,
Geo. J. Friedrichs,
per pro. A. G. Friedrichs.

Witnesses:
J. Blanc Monroe
Chas. F. Hester.

C. C. FRIEDRICHS,
Notary Public.

I, J. Ward Gurley, District Attorney for the Parish of Orleans, State of Louisiana, do hereby certify that I have examined the foregoing Act of Incorporation of the Louisiana State Medical Society, and am of opinion that the objects and purposes of said corporation, as specified in said act, are legal, and that none of the provisions therein contained are contrary to law.

New Orleans, La., June 5, 1903.

J. WARD GURLEY,
District Attorney for Parish of Orleans.

I, the undersigned Recorder of Mortgages, in and for the Parish of Orleans, State of Louisiana, do hereby certify that the above and foregoing Act of Incorporation of the LOUISIANA STATE MEDICAL SOCIETY, and the certificate of the District Attorney for the Parish of Orleans, was this day recorded in my office, in book 750, folio 432, June 10, 1903.

(Signed) EMILE LEONARD, D. R.

CONSTITUTION

ARTICLE I

Name of Society

The name and title of this organization shall be the LOUISIANA STATE MEDICAL SOCIETY.

ARTICLE II

Purposes of the Society

The purposes of this Society shall be to federate and bring into one compact organization the eligible members of the medical profession of the State of Louisiana and unite with similar associations in other States to form the American Medical Association, with a view to the extension of medical knowledge and to the advancement of medical science; to the elevation of the standard of medical education, and to the enactment and enforcement of just medical laws; to the promotion of friendly intercourse among physicians, and to the guarding and fostering their material interests, and to the enlightenment and direction of public opinion in regard to the great problems of State medicine; so that the profession shall become more capable and honorable within itself and more useful to the public, in the prevention and cure of disease, and in prolonging and adding comfort to life.

ARTICLE III

Component Societies

Component Societies shall consist of those District and Parish Medical Societies which hold charters from this Society.

ARTICLE IV

Composition of the Society

This Society shall consist of active, associate, honorary, inactive and intern members.

Section 1. *Active Members*—The active members of this Society shall be the active members of the component district and parish medical societies, also physicians otherwise eligible to active membership in any district or parish not having a component society; provided, that after the organization in such district or parish of a component society they shall become members of said society or forfeit membership in the State Society.

Section 2. *Associate Members*—Associate members of component societies will be eligible for associate membership; also physicians in the service of the Veterans' Bureau, Army, Navy or Public Health Service of the United States, who are on active duty and stationed in the state may be elected to associate membership when endorsed by two active members in good standing of the Society to whom they are personally known. Such membership is not to have the privileges of executive character or protection of medical defense. Dues for associate membership will be one-half amount of dues for active membership.

Section 3. *Honorary Members*—Any member in good standing who has rendered meritorious service to organized medicine or contributed to the advancement of scientific medicine, or any person not a member distinguished in medicine or the collateral branches of science may, upon recommendation of the Council, be elected to honorary membership. Active members elected to honorary membership shall have the same rights and privileges of active members but shall not be permitted to hold office. Dues and assessments of such members shall be deleted. Honorary membership in a component society does not necessarily imply honorary membership in the State Society.

Section 4. *Inactive Members*—Inactive membership shall include physicians who have been in active practice and members of long standing of the State Society who shall be approved for such membership by the Executive Committee. They shall not be entitled to medical defense nor shall they be eligible to hold office. No dues shall be required of inactive members.

Section 5. *Intern Members*—Physicians serving internships or residencies in accredited hospitals shall during their period of internship or residency be eligible to membership in this Society through their respective component societies as intern members. They shall not be entitled to medical defense, nor shall they be eligible to hold office or vote. Dues for intern members shall be $1.00 per year.

Section 6. *Parish and District Societies*—Parish and District Societies cannot include in their membership physicians who are not members in good standing of the Louisiana State Medical Society. Component District and Parish Societies not complying with this provision shall forfeit their Charters.

ARTICLE V

Guests

Any distinguished person, medical or otherwise may become a Guest during any annual session upon invitation of the Society or its Executive Committee, and shall be accorded the privilege of participating in all of the scientific program for that session.

ARTICLE VI

Delegates

Delegates shall be those members who are elected in accordance with this Constitution and By-Laws to represent their respective component district and parish societies in the House of Delegates of this Society.

ARTICLE VII

House of Delegates

The House of Delegates shall be the legislative and business body of the Society, and shall consist of (1) delegates elected by the component district or parish societies, and (2) the officers of the Society as defined in this Constitution and (3) an Advisory Body composed of Past Presidents without the power of voting on Constitutional offices.

ARTICLE VIII

District Societies

The Executive Committee shall provide for the organization of district and parish societies for the promotion of the best interests of the profession.

ARTICLE IX

Sessions and Meetings

Section 1. The Society shall hold an annual session, during which there shall be held daily scientific and general meetings which shall be open to all registered members and guests. The time, character and number shall be determined by the Executive Committee.

Section 2. The place for holding each annual session shall be fixed by the House of Delegates. In case of emergency and a meeting can not be held at a designated place and it is impossible to hold a meeting of the House of Delegates, the Executive Committee will be empowered to alter place of meeting.

Section 3. The Executive Committee shall provide for a division of the scientific program of the Society into appropriate sections at its first meeting, which will take place not later than thirty days after the last day of the annual session, and the President shall appoint chairmen of the various sections not later than thirty days following his installation.

ARTICLE X

Officers

Section 1. The officers of this Society shall be a President, President-elect, three Vice-Presidents, a Secretary-Treasurer, and Councilors, one from each Congressional District, the Chairman and the Vice-Chairman of the House of Delegates and the retiring President of the Society.

Section 2. The President, President-elect, Vice-President, Chairman and Vice-Chairman of the House of Delegates shall be elected for a term of one year. The Secretary-Treasurer shall be elected for a term of not more than five years. The Councilors shall be elected for a term of two years each, being divided into two groups so that as nearly as possible half shall be elected for one year and the rest for two years, and thereafter each group for two years. All of these officers shall serve until their successors are elected and installed.

Section 3. The officers of this Society and the names of the members to be presented to the Governor of the State for appointment on the State Board of Medical Examiners shall be elected by the House of Delegates on the morning of the last day of the annual session, and a person shall not be elected to any office or be recommended for appointment on the State Board of Medical Examiners, who is not in attendance (except he has been a faithful attendant at the annual sessions and has a valid excuse) and who has not been a member of the Society for the past two years.

ARTICLE XI

Funds and Expenses

Funds for meeting the expenses of the Society shall be arranged for by the collection of annual dues, by voluntary contributions, assessments and public benefits, and from the sale of the publications and by any other means sanctioned by the Executive Committee and approved by the Society. Funds may be appropriated to defray the expenses of the Annual Session, for publication, and for such other purposes as will promote the welfare of the Society and profession.

ARTICLE XII

Referendum and Recall

Section 1. The General Meeting of the Society may, by a two-thirds vote, order a general referendum upon any question pending before the House of Delegates, and the House of Delegates may, by similar vote of its own members, or after a like vote of the General Meeting, submit any such question to the membership of the Society for a final vote; and if the persons voting shall comprise a majority of all the members present, a majority of such vote shall determine the question, and be binding upon the House of Delegates.

Section 2. Any officer neglecting his duty or violating any article of this Charter, Constitution or By-Laws may be removed from his office after due trial at any regular or special session of the House of Delegates, provided thirty days' notice be furnished in writing to the officer so charged. The charges should originate in the Council either directly or by the request of twenty-five active members in good standing.

ARTICLE XIII

The Seal

The Society shall have a common seal, with power to break, change or renew the same at pleasure.

ARTICLE XIV

Amendments

The House of Delegates may amend any article of this Constitution by a two-thirds vote of the delegates registered at that annual session, provided that such amendments shall have been presented at a previous annual meeting and have been approved by the House of Delegates.

BY-LAWS

CHAPTER I

Membership

Section 1. *Privileges*—All members in good standing shall be privileged to attend all meetings and take part in all proceedings of the annual session, but only active members shall be eligible to any office within the gift of the Society.

Section 2. *Rights*—The name of a physician upon the properly certified roster of members of a component district or parish shall be prima facie evidence of his right to register at the annual sessions of this Society provided his State Society dues have been received for the current year.

Section 3. *Suspension or Expulsion*—No person who is under sentence of suspension or expulsion from any component society of this Society, or whose name has been dropped from its roll of members, shall be entitled to any of the rights or benefits of this Society, nor shall he be permitted to take any part in any of its proceedings until such time as he has been relieved of such disability.

Section 4. *Registration*—Each member in good standing in attendance at the annual session shall, after registering, receive a badge which shall be evidence of his right to all privileges of membership at that session. No member or delegate shall take part in any of the proceedings of an annual session until he has complied with the provisions of this section.

CHAPTER II

Annual and Special Sessions of the Society

Section 1. *Annual Sessions*—The Society shall hold an annual session at such time and place as has been fixed at the preceding annual session or as fixed by this Constitution and By-Laws. At this annual session shall be held general, installation and scientific meetings and the annual meeting of the House of Delegates.

Section 2. *Special Sessions*—Special sessions of either the Society or House of Delegates shall be called by the President at his discretion or by petition of twenty-five active members.

Section 3. *Meetings*—The *General Meetings* shall include all registered active members who shall have equal rights to participate in the proceedings and discussions and to vote on pending questions. These meetings shall be for the purpose of considering matters affecting the welfare of the Society and the profession. The entire time of the session shall be devoted to business of interest to the Society and the profession. Each General Meeting shall be presided over by the President, or in his absence or disability, or by his request, by one of the Vice-Presidents. A general meeting shall be called at the conclusion of the annual session of the House of Delegates for the purpose of considering the report of the House of Delegates. The General Meeting shall have authority to create committees or commissions for scientific investigation of special interest and importance to the profession and public, and to receive and dispose of reports of the same; but any expense in connection therewith must first be approved by the House of Delegates.

An *Installation Meeting* shall be held during the annual session. The nature of this meeting shall be left to the discretion of the Chairman of the Committee on Arrangements. At this meeting the reports of the retiring president and the address of the incoming president, and the annual oration man be rendered.

The *Scientific Meetings* shall be devoted to papers and discussions relating to scientific medicine, arranged by the Committee on Scientific Work. The scientific program of each meeting shall be divided into two principal sub-divisions, namely, Medicine and its allied branches, and Surgery and its allied branches. These two main sub-divisions shall be further sub-divided into sections, each section having a specified number of papers, as follows:

Medicine and Allied Branches	
Medicine and Therapeutics	7
Pediatrics	3
Nervous Diseases	3
Bacteriology and Pathology	3
Public Health and Sanitation	5

Gastro-enterology	2
	(23)
Surgery and Allied Branches:	
General Surgery (Including Dermatology)	7
Gynecology and Obstetrics	4
Eye, Ear, Nose and Throat, Including Stomatology	5
Urology	2
Radiology	3
Orthopedic Surgery	2
	(23)

The order of exercises, papers and discussions as set forth in the official program shall be followed from day to day until it has been completed; but the papers of one session shall not encroach upon the papers of another session. No address or paper before the Society, except those of the President and orators, shall occupy more than fifteen minutes in its delivery; and no member shall offer discussion longer than five minutes, nor more than once on any subject. All papers read before the Society shall be its property. Each paper shall be deposited with the Secretary-Treasurer when read, and if this is not done the author shall not be allowed to read a paper at the two next succeeding Annual Meetings. Each paper shall be read by its author. If this condition is not complied with the author shall not be allowed to read a paper at the two next succeeding Annual Meetings. Any author failing to read his paper at any Annual Meeting shall not be allowed to read a paper at the two next succeeding Annual Meetings. If any author shows his good intent by forwarding his paper to the Secretary-Treasurer in time for the Annual Session, he shall not suffer this penalty. Any author suffering any of the above penalties shall also not be allowed to appear on the official program in the capacity of opening discussions for a like time. No member shall present more than one paper at any Annual Meeting.

The House of Delegates shall meet annually at the time and place of the Annual Session of the Society.

CHAPTER III

Installation of President

The President-elect shall be installed as President during the annual session, the time and place to be left to the discretion of the Chairman of the Committee on Arrangements. In the event that a scheduled annual session can not be held the Executive Committee shall determine time and place for installation.

CHAPTER IV

House of Delegates

Section 1. *Meetings*—The House of Delegates shall so fix its hours of meeting as not to conflict with the General Meeting of the Society, or with the meeting held for the address of the President and the annual oration, and so as to give delegates an opportunity to attend the other scientific proceedings and discussions so far as is consistent with their duties. The House of Delegates shall meet on the morning of the day previous to the Annual Meeting, and it may remain in session up to the final adjournment of the General Meeting. On the morning of the last day of the session the House of Delegates shall elect its Chairman and Vice-Chairman for the ensuing year.

Section 2. *Number of Delegates from Each Component Society*—Each Component Society shall be entitled to send to the House of Delegates each year one delegate for every twenty-five active members, and one for each fraction thereof, but each District and Parish Society holding a charter from this Society, which has made its annual report shall be entitled to its representation. Each district society shall be entitled to only one delegate.

Section 3. *Quorum*—A majority of the registered delegates shall constitute a quorum, and all of the meetings of the House of Delegates shall be open to members of the Society.

Section 4. *Aims and Purposes*—It shall, through its officers, Executive Committee and otherwise, give diligent attention to and foster the scientific work and spirit of the Society, and shall constantly study and strive to make each Annual Session a stepping stone to future ones of higher interest. It shall consider and advise as to the material interests of the profession, and of the public in those important matters wherein it is dependent upon the profession, and shall use its influence to secure and enforce all proper medical and public health legislation, and to diffuse popular information in relation thereto. It shall make careful inquiry into the condition of the profession of each parish of the State, and shall have authority to adopt such methods as may be deemed most efficient for building up and increasing the interest in such parish societies as already exist, and for organizing the profession in parishes where societies do not exist. It shall encourage postgraduate work in medical centers, as well as home study and research, and shall endeavor to have the results of the same utilized and intelligently discussed in the component societies.

Section 5. *Delegates to A. M. A.*—It shall elect representatives to the House of Delegates of the American Medical Association in accordance with the Constitution and By-Laws of that body in such a manner that not more than one-half of the delegates shall be elected in any one year, when possible.

Section 6. *Charters*—It shall, upon application, provide and issue charters to district and parish societies organized to conform to the spirit of this Constitution and By-Laws.

Section 7. *Organization*—In sparsely settled sections it shall have authority to organize the physicians of two or more parishes into societies to be designated by hyphenating the names of two or more parishes so as to distinguish them from district and other classes of societies, and these societies, when organized and chartered, shall be entitled to all the privileges and representation provided herein for parish societies until such parishes may be organized.

Section 8. *District Societies*—In extraordinary cases it may form district societies of groups of parishes or parts of parishes for the purpose of fostering the scientific interest of the members, but this is not in any sense to be construed as interfering in organizing the various parishes into component societies for the purpose of representation in the House of Delegates.

Section 9. *Committees*—It shall have authority to appoint committees for special purposes from among members of the Society who are not members of the House of Delegates, and such committees may report to the House of Delegates in person, and may participate in the debate thereon.

Section 10. *Memorials and Resolutions*—It shall approve all memorials and resolutions issued in the name of the Society before the same shall become effective.

Section 11. *Publication of Proceedings*—It shall present a summary of its proceedings to the last General Meeting of each Annual Session, and publish the same in the Transactions or Offical Journal.

CHAPTER V

Fiscal Year

The Fiscal year of the Society shall begin January first.

CHAPTER VI

Election of Officers

Section 1. Nominations shall be made in open meeting of the House of Delegates on the first day of its meeting and the election made by secret ballot on the last day, and a majority of the votes cast shall be necessary to elect.

Section 2. *Dual Office-Holding*—No member may hold two elective offices simultaneously except those elected to standing committees.

CHAPTER VII

Duties of Officers

Section 1. *President*—The President shall preside at all meetings of the Society; shall appoint all committees not otherwise provided for; shall deliver an annual address at such time as may be arranged; shall give a deciding vote in case of a tie, and shall perform such other duties as custom and parliamentary usage may require. He shall be the real head of the profession in the State during his term of office, and, as far as practicable, shall visit by appointment the various sections of the State and assist the Councilors in building up the district and parish societies, and in making their work more practical and useful.

Section 2. *Vice-Presidents*—The Vice-Presidents shall assist the President in the discharge of his duties.

Section 3. *President-elect*—The President-elect shall familiarize himself with the affairs of the Society, keeping in touch with the organization, and shall assume the duties of President upon his installation.

Section 4. *Chairman of House of Delegates*—The Chairman of the House of Delegates shall preside at meetings of the House of Delegates and shall perform such duties as custom and parliamentary usage require.

Section 5. *Vice-Chairman of House of Delegates*—The Vice-Chairman of the House of Delegates shall officiate for the Chairman in his absence or at his request.

Section 6. *Secretary-Treasurer*—The Secretary-Treasurer, as Chairman of the Committee on Scientific Work, shall prepare and issue the programs for and attend all meetings of the Society, and he shall keep minutes of their respective proceedings in separate record books. He shall be custodian of all record books and papers belonging to the Society. He shall provide for the registration of the members and delegates at the Annual Session. He shall keep a card index register of all the legal practitioners of the State by parishes, noting on each his status in relation to his society, and upon request shall transmit a copy of this list to the American Medical Association for publication. In so far as it is in his power he shall use the printed matter, correspondence and influence of his office to aid the Councilors in the organization and improvement of the parish societies, and in the extension of the power and usefulness of this Society. He shall conduct the official correspondence, notifying members of meetings, officers of their election and committees of their appointment and duties. The Secretary-Treasurer shall give bond for the trust reposed in him as fixed by the Executive Committee. He shall demand and receive all funds due the Society, together with the bequests and donations. He shall, in general, have the care and management of the fiscal affairs of the Society. He shall be allowed to draw checks for the purpose of meeting and paying of the routine expenses of the Society without vouchers from the President, provided such amounts shall not be in excess of the amount specified by the Budget and Finance Committee; he shall submit the books of the Society to such examinations as the House of Delegates may order, and he shall at the first session of the annual meeting of the House of Delegates render an account of his activities and of the financial status of the organization. He shall collect and enter upon the books the annual dues for members and perform such other duties as may pertain to his office. In order that the Secretary-

Treasurer may be enabled to give that amount of time to his duties which will permit of his becoming proficient, it is desirable that he should receive compensation. The amount of salary shall be fixed by the Executive Committee within limits fixed by Budget and Finance Committee.

CHAPTER VIII

Council

Section 1. *Meetings*—The Council shall hold meetings during the Annual Session of the Society at such times as necessity may require, subject to the call of the Chairman or on petition of three Councilors. It shall meet on the date of the first meeting of the Executive Committee after the Annual meeting of the House of Delegates for reorganization and for the outlining of work for the ensuing year. At this meeting it shall elect a Chairman and Secretary and it shall keep a permanent record of its proceedings. It shall, through its Chairman, make an annual report to the House of Delegates at such time as may be provided.

Section 2. *Duties of Councilors*—Each Councilor shall be the organizer, peacemaker and censor for his district. He shall visit each parish in his district at least once a year for the purpose of organizing component societies where none exist, for inquiring into the condition of the profession, and for improving and increasing the zeal of the parish societies and their members. He shall make a report including activities of the component societies in his district, and of the condition of the profession of each parish in his district, to each Annual Session of the House of Delegates. The necessary traveling expenses incurred by such Councilor in the line of the duties herein imposed may be allowed by the House of Delegates upon a proper itemized statement, but this shall not be construed to include his expense in attending the Annual Session of the Society.

Section 3. *Duties of Council*—Collectively the Council shall be the Board of Censors of the Society. It shall consider all questions involving the rights and standing of members, whether in relation to other members, to the component societies, or to this Society. All questions of an ethical nature brought before the House of Delegates or the General Meeting shall be referred to the Council without discussion. It shall hear and decide all questions of discipline affecting the conduct of members or of a district or parish society, upon which an appeal is taken from the decision of an individual Councilor. Its decision in all such cases shall be final.

Section 4. *Rights*—The Council shall have the right to communicate the view of the profession and of the Society in regard to health, sanitation and other important matters to the public and the lay press. Such communications shall be officially signed by the Chairman and Secretary of the Council as such. Councilors shall be ex-officio members of the House of Delegates.

Section 5. *Reports*—If a Councilor fails to have a written report of his Councilor District at the Annual Meeting, his position shall be declared vacant, if so decided by a majority vote of the House of Delegates.

CHAPTER IX

Committees

Standing Committees shall be as follows:

Committee on Arrangement
Committee on Budget and Finance
Committee on Congressional Matters
Committee on Journal
Committee on Maternal Welfare
Committee on Medical Defense
Committee on Medical Education
Committee on Medical Testimony
Committee on Public Policy and Legislation
Committee on Scientific Work

Such committees shall be elected by the House of Delegates unless otherwise provided.

Section 1. *Committee on Arrangement*—The Committee on Arrangement shall consist of the component society in the area in which the Annual Session is to be held. It shall, by committees of its own selection, provide suitable accommodations for the meeting places of the Society and of the House of Delegates, and of their respective committees, and shall have general charge of all the arrangements. Its Chairman shall report an outline of the arrangements to the Secretary-Treasurer for publication in the program and Official Journal, and both shall make additional announcements during the session as occasion may require.

Section 2. *Committee on Budget and Finance*—A Committee on Budget and Finance, whose duty shall be to present a budget of income and expense for the ensuing year. It also shall audit the books of the Secretary-Treasurer and of all committee funds and perform such advisory duties as may be of benefit to the Society, and shall make its report to the Executive Committee at the first meeting and also to the House of Delegates. It shall consist of three members, appointed by the Chairman of the House of Delegates, with overlapping terms of three years. At its discretion the services of an accredited public accountant may be employed.

Section 3. *Committee on Congressional Matters*—The Committee on Congressional Matters shall consist of three members, appointed by the President for a term of one year. The duties of this committee shall be to keep in touch with congressional bills and legislative matters which pertain to medicine and public health and inform members of the Society concerning status of same.

Section 4. *Committee on Journal*—The Journal Committee shall consist of five members with overlapping terms of three years. At the expiration of the respective terms their successors shall be elected by the House of Delegates each for a term of three years. In the event of a vacancy occurring by resignation, death or other causes, the President shall appoint successors to serve until the next meeting of this Society, successors being then elected by the House of Delegates. The Journal Committee shall have full control of the editing and publishing of the New Orleans Medical and Surgical Journal. The State Society shall pay the Journal seventy-five cents ($0.75) per year per member in consideration of which each member shall receive the Journal for one year. The Journal Committee shall have charge of the publication of transactions, papers, and other matters.

Section 5. *Committee on Maternal Welfare*—The Committee on Maternal Welfare shall consist of eight members appointed by the President, one from each Congressional District. They shall consider all matters pertaining to maternal and child welfare which concern the public, members of this Society and the State health department. They shall cooperate with various agencies concerned with this work and determine policies for the benefit of the public and of the medical profession.

Section 6. *Committee on Medical Defense*—The administration of Medical Defense of this Society shall be entrusted to a standing committee of three members with overlapping terms to be elected by the House of Delegates; one member to be elected at each annual session for a term of three years. This committee shall have full authority governing all matters pertaining to the Medical Defense of this Society; with power to employ counsel, summon and employ expert witnesses and incur such other expenses as in the judgment of the committee may be necessary in the defense of members against whom suits may be brought; provided, always, that the total expenditure of any single suit shall not exceed 25 per cent of the fund available at the time suit is incurred. The Medical Defense Committee shall make an annual report to the House of Delegates of the cases in which it has been of service to members, and furnish an account of money received and expended, such report to be published in the official journal of the Louisiana State Medical Society at the time and in the manner that reports of other committees of the Society are published. The financial report of the Committee shall be submitted to and approved by the Committee on Budget and Finance. Each member of the Committee on Medical Defense of this Society shall be entitled to an honorarium of $10.00 per diem with traveling expenses, if required to go out of town in the investigation of any case or in attendance at court, and these same fees shall be allowed to expert witnesses under similar circumstances. The Committee on Medical Defense shall have power to adopt such other rules, not in conflict with the foregoing, as in their judgment may seem necessary until the next Annual Meeting of the House of Delegates, when such rules shall take the regular course of amendments.

Section 7. *Committee on Medical Education*—A Committee on Medical Education consisting of three members appointed by the President with overlapping terms of three years, whose duty shall be to furnish a report to the House of Delegates upon the status of medical education in the State, and to work in accordance with the Council on Medical Education and Hospitals of the American Medical Association. This appointment shall be made not later than thirty days after the last day of the Annual Session.

Section 8. *Committee on Medical Testimony*—The Committee on Medical Testimony shall consist of five members appointed by the President with overlapping terms of three years. They shall make recommendations for the improvement of expert testimony by the medical profession in accordance with legal regulations. They shall be empowered to investigate all complaints relative to violations in medical or expert testimony and to submit recommendations for improvement of legislation governing expert testimony.

Section 9. *Committee on Public Policy and Legislation*—The Committee on Public Policy and Legislation shall consist of three members and the President and Secretary-Treasurer. Under the direction of the House of Delegates it shall represent the Society in securing and endorsing legislation in the interest of the public health and of scientific medicine. It shall keep in touch with professional and public opinion, shall endeavor to shape legislation so as to secure the best results for the whole people, and shall utilize every organized influence of the profession to promote the general influence in local, State and National affairs and elections. Its work shall be done with the dignity becoming a great profession and with that wisdom which will make effective its power and influence. It shall have authority to be heard before the entire Society upon questions of great concern at such time as may be arranged during the Annual Session. The President shall have power to enlarge the Committee as exigency requires.

Section 10. *Committee on Scientific Work*—The Committee on Scientific Work shall consist of three members, of which the Secretary-Treasurer shall be Chairman, and shall determine the character and scope of the scientific proceedings of the Society for each session, subject to the approval of the Executive Committee. Early in January it shall publish in the official Journal the names of the Chairmen of Sections. The Chairman of each Section shall present the complete program of his Section to this Committee not later than 60 days previous to the Annual Session. Two weeks previous to each Annual Session it shall issue a program announcing the order in which papers, discussion and other business shall be presented, which shall be adhered to by the Society as nearly as practicable.

CHAPTER X

Medical Defense

Section 1. Active members of the Louisiana State Medical Society who have paid all dues, assessments and other charges assessed or levied by the Louisiana State Medical Society, shall be entitled, on conditions hereinafter specified, to receive without personal expense therefor, legal advice and court service of an attorney or attorneys at law in the employ of the Society, or others selected by the Committee on Medical Defense, in consultation with the defendant; and witness fees for the purpose of conducting their defense in any courts of the State in which the suit may be filed or threatened to be filed.

Section 2. Fifty cents per capita shall be set aside as a special fund for Medical Defense. Whenever such fund shall exceed the sum of $20,000, the surplus over and above this amount may be used for such other purposes as the House of Delegates may direct.

Section 3. The liability of this Society shall include in Medical Defense only the expenses necessary for the legal defense of its members and not damages awarded.

Section 4. The Society shall not undertake the defense of a member in a suit that may be brought to secure indemnity for service rendered prior to January 1, 1914, nor in any case in which the member who applies for medical defense by the Society, has failed to pay his annual dues for 1914 or prior to the rendering of services which are the basis of the suit; and medical defense by the Society shall not be available to those who are delinquent or to those who have not paid the annual dues of the Society prior to the rendering of services for which indemnity is asked.

Section 5. A member desiring to avail himself of the services of the Committee on Medical Defense in connection with litigation brought or threatened must first submit to a local committee of his parish medical society—to be composed of the President, Secretary and one other member in good standing, who may be nominated by the defendant—a full statement of the question at issue, including the diagnosis and treatment of the case and the names of physicians, nurses and other persons having knowledge of the same, who may be summoned as witnesses.

Section 6. The committee of the parish medical society shall immediately, after an investigation of all the circumstances and facts, transmit its report, with recommendations, to the Committee on Medical Defense of this Society.

Section 7. Accompanying such report from the parish society, if favoring medical defense by the Society, there must also be furnished the written authority of the defendant granting to the Medical Defense Committee of this Society full power to act in his behalf, and an agreement that his case shall not be compromised or settled without the consent of a majority of the Committee on Medical Defense.

Section 8. In the event that the parish committee shall fail to recommend the case as one worthy of the recognition of this Society, a direct appeal may be made to the Committee on Medical Defense of this Society, whose decision shall be final.

Section 9. Suits brought against the estate of a deceased member shall be defended as if that member were alive; provided, that such member was in good standing in the Society at the time of his death and that services for which indemnity is asked were rendered while the deceased was a member in good standing.

Section 10. The defense promised in Chapter X shall apply when the suit is brought by the patient, by the nurse or anyone who claims to have been injured by an act of the doctor at the time the doctor was in the actual performance of his professional duty.

CHAPTER XI

Assessments and Expenditures

Section 1. *Assessments and How Payable*—Each active member shall pay into the Society an annual assessment of twenty-five ($25.00), which shall constitute his membership dues. Those specifically exempted in the Constitution shall not be liable for this assessment. Intern members shall pay the sum of one dollar ($1.00) per annum. Of the amount collected from active members, one dollar ($1.00) per capita shall be set aside as an annual entertainment fund. In addition, one half dollar (50c) per capita shall be paid from the dues collected from active members into the Medical Defense Fund, one dollar ($1.00) into the Indigent Physicians Fund and one half dollar (50c) into the fund of the Committee on Public Policy and Legislation. After the first day of July and before the first day of October new active members shall pay one-half of the annual dues for the fiscal year; after the first day of October of the current year new members admitted to the Society shall pay one-quarter of the annual dues for the fiscal year.

Section 2. Component societies having a classification of Intern Membership shall pay dues to the State Society for each member in the amount of one dollar ($1.00) per annum.

Section 3. Dues must be paid in advance. The Secretary of each component society must forward to the Secretary-Treasurer of the State Society the dues and assessments of each member of his Society.

Section 4. *Delinquencies*—If dues and assessments are not paid by the date of the annual meeting a member shall be suspended. Suspended members shall not be eligible to benefits.

Section 5. Physicians in organized parishes with the proper qualifications can send their dues direct to the State Society when the Parish Society in which parish they reside becomes inactive.

Section 6. *Mode of Appropriating Money*—All motions or resolutions appropriating money shall specify a definite amount, or so much thereof as may be necessary for the purpose indicated, and must be endorsed by the Budget and Finance Committee and submitted to the House of Delegates for approval.

CHAPTER XII

Travel Expense

Travel expense of members of the Executive Committee to meetings of that committee; expense of delegates to meetings of the House of Delegates of the American Medical Association; and expense of members on approved committee assignments, shall be paid from the general fund upon receipt of statement of expenditure.

CHAPTER XIII

Rules of Conduct

The principles set forth in the Code of Ethics of the American Medical Association shall govern the conduct of members in their relation to each other and to the public.

CHAPTER XIV

Rules of Order

The deliberations of this Society shall be governed by parliamentary usage as contained in Robert's Rules of Order, unless otherwise determined by a vote of its respective bodies.

CHAPTER XV

Parish Societies

Section 1. *How Created*—All parish societies now in affiliation with the State Society or those that may hereafter be organized in this State, which have adopted principles of organization not in conflict with this Constitution and By-Laws, shall, upon application to the Executive Committee, receive a charter from and become a component part of this Society.

Section 2. *Mode of Organization*—As rapidly as can be done after the adoption of this Constitution and By-Laws, a medical society shall be organized in every parish in the State in which no component society exists, and charters shall be issued thereto.

Section 3. *Charters*—Charters shall be issued only upon approval of the Executive Committee and shall be signed by the President and Secretary-Treasurer, Chairman of the Council and the Councilor of the district. The Executive Committee shall have authority to revoke the charter of any component district or parish society whose actions are in conflict with the letter or spirit of this Constitution and By-Laws.

Section 4. *Only One Charter to Each Parish Permitted*—Only one component medical society shall be chartered in any parish. Where more than one parish society exists, friendly overtures and concessions shall be made, with the aid of the Councilor of the District, if necessary, and all of the members brought into one organization. In case of failure to unite, an appeal may be made to the Council, which shall decide what action shall be taken.

Section 5. *Qualifications of Members*—Each parish society shall judge of the qualifications of its own members, but, as such societies are the only portals to this Society, and to the American Medical Association, every reputable and legally registered physician who is practicing, or who will agree to practice non-sectarian medicine, shall be eligible for membership. Before a charter is issued to any parish society, full and ample notice and opportunity shall be given to every such physician in the parish to become a member.

Section 6. *Right of Appeal*—Any physician who may feel aggrieved by the action of the society of his parish in refusing him membership or in suspending or expelling him, shall have the right of appeal to the Council and to the House of Delegates.

Section 7. *Hearing of Appeals by Council*—In hearing appeals the Council may admit oral or written evidence as in its judgment will best and most fairly present the facts, but in the case of every appeal, both as a Board and as individual Councilors in district and parish work, efforts at conciliation and compromise shall precede all such hearings.

Section 8. *Transfer of Membership*—When a member in good standing in a component society moves to another parish in this State, his name, upon request, shall be transferred without cost to the roster of the parish society into whose jurisdiction he moves, subject to the rules governing such society.

Section 9. *Choice of Membership*—A physician living on or near a parish line may hold his membership in that parish most convenient for him to attend, on permission of the society in whose jurisdiction he resides.

Section 10. *Rights of Component Societies*—Each parish society shall have general direction of the affairs of the profession in the parish, and its influence shall be constantly exerted for bettering the scientific, moral and material condition of every physician in the parish; and scientific efforts shall be made by each member, and by the society as a whole, to increase the membership until it embraces every qualified physician in the parish. In any unorganized parish in which there are ten or more members of the State Society, these members must effect organization or forfeit their membership in the State Society.

Section 11. *Meetings*—Frequent meetings shall be encouraged, and the most attractive programs arranged that are possible. The younger members shall be especially encouraged to do postgraduate and original research work, and to give the Society the first benefit of such labors.

Section 12. *Delegates*—At some meeting in advance of the fiscal year of this Society each parish society shall elect a delegate or delegates, and alternate or alternates, to represent it in the House of Delegates of this Society, in the proportion of one delegate and alternate to each twenty-five active members, or fraction thereof, and the secretary of the society shall send a list of such delegates and alternates to the Secretary-Treasurer of this Society. Should any delegate or alternate so elected to represent any parish society fail to attend any meeting, it shall be the duty of the president of such parish medical society, or in his absence, the vice-president thereof, to appoint an alternate or alternates in writing or by telegraph. Component societies failing to comply with this provision shall be denied representation in the House of Delegates, unless otherwise decided by a two-thirds majority of delegates at meeting when a request for different action is made.

Section 13. *Duties of Secretaries of Component Societies*—The secretary of each parish society shall keep a roster of its members, and a list of the non-affiliated registered physicians of the parish, in which shall be shown the full name, address, college and date of license to practice in this State, and such other information as may be deemed necessary. He shall furnish an official report containing such information upon blanks supplied him for the purpose, to the Secretary of this Society. In keeping such roster the secretary shall note any changes in the personnel of the profession by death, or by removal to or from the parish, and in making his annual report he shall be certain to account for every physician who has lived in the parish during the year.

CHAPTER XVI

Amendments

These By-Laws may be amended at any Annual Session by a majority vote of all the delegates present at that session, after the amendment has laid upon the table one day or by unanimous consent. A copy printed on gummed paper should be mailed to each member within sixty (60) days of its enactment.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

NEWS ITEMS

The 1943 medical records prize was awarded to Dr. W. D. Beacham at the American College of Surgeons meeting recently held in Cleveland.

Drs. Wm. H. Gillentine and Thomas Benton Sellers were guest speakers at the meeting of the South Mississippi Medical Society on December 12 at Hattiesburg. Dr. Gillentine spoke on "Cure of Heart Disease" and Dr. Sellers on "Management of the Displaced Uterus".

Dr. Howard Mahorner attended the meeting of tre Southern Surgical Association in Hot Springs, Va., December 10-12.

At the November meeting of the EENT section of the Charity Hospitay Visiting Staff the following scientific program was presented:

Presentation of Interesting Eye Cases by Dr. Charles Bahn.

Relation of Poliomyelitis and ENT Conditions by Dr. Branch J. Aymond.

Dr. F. L. Jaubert was recently re-elected president of the Louisiana Society for Crippled Children.

Dr. Henry D. Ogden attended the third annual meeting of the American Academy of Allergy in New York, November 25-27. A paper on "Severe Allergic Reactions During Cystoscopy" was prepared by Drs. Ogden, Harry L. Zengel, Jr., and John F. Lally for presentation at the meeting.

Dr. John M. Whitney was elected secretary of the Southern Branch of the American Public Health Association at their meeting in Miami recently.

Dr. Edwin L. Zander was recently elected a member of the board of directors of the New Orleans Kiwanis Club.

HOSPITAL NEWS

At a recent meeting of the Medical Advisory Committee to the Charity Hospital Staff Dr. Edwin L. Zander was elected president. Other officers elected were as follows: Dr. E. Garland Walls, vice-president; Dr. N. Polmer, secretary; Drs. Edgar Hull, John Oakley, N. J. Tessitore and Julius Lane Wilson, members of the Committee.

Dr. N. J. Tessitore was elected chairman; Drs. Edgar Hull and E. Garland Walls, members of the Qualifications Committee of the Charity Hospital.

Physicians elected to the Medical Division of the Charity Hospital Visiting Staff are as follows: Dr. Wm. A. Sodeman, chairman; Dr. Edwin S. Kagy, vice-chairman; and Dr. Joseph W. Wells, secretary.

Newly elected members of the Surgical Division of the Charity Hospital Visiting Staff are as follows: Dr. Frank Loria, chairman; Dr. Daniel W. Goldman, vice-chairman; and Dr. Edward Nelson, secretary.

Physicians elected to the sectional sections of the Charity Hospital Visiting Staff are as follows:

Surgery—Dr. L. J. O'Neil, chairman; Dr. Alex Powe, vice-chairman; and Dr. M. L. Stadiem, secretary.

Ob.-Gyn.—Dr. Conrad Collins, chairman; Dr. D. W. Goldman, vice-chairman; and Dr. Claude Callendar.

E. E. N. T.—Dr. Charles A. Bahn, chairman; Dr. Paul Lastrapes, secretary.

Urology—Dr. John G. Pratt, chairman; Dr. R. J. Mailhes, vice-chairman; and Dr. John G. Menville, secretary.

Orthopedic—Dr. Harry Morris, chairman; Dr. Irvin Cahen, vice-chairman; and Dr. A. R. Altenberg, secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

IMPORTANT

There is urgent need in the office of the Journal for additional copies of the November, 1946 issue. We would ask members of the State Society to forward their copies to us when they are through with them.

RAPIDES PARISH MEDICAL SOCIETY

The following is a list of the officers elected by the Rapides Parish Medical Society for 1947.

Dr. H. P. Forsythe, President, Alexandria, La.

Dr. H. C. Gahagan, 1st Vice President, Alexandria, La.

Dr. Morris Hair, 2nd Vice President, Lecompte, La.

Dr. John S. Rozier, Secretary-Treasurer, Alexandria, La.

Board of Censors: Dr. H. Aubrey White, Alexandria, La.

House of Delegates: Dr. M. B. Pearce, Alexandria, La. Alternate: Dr. H. A. White, Alexandria, La.

John S. Rozier, M. D.,
Secretary-Treasurer.

NATIONAL CONFERENCE ON MEDICAL SERVICE

There will be held in Chicago on Sunday, February 9, the 20th Annual Meeting of the National Conference on Medical Service. This will be an all-day session which will be opened by the address of the President, Dr. Cleon A. Nafe of Indianapolis. There will be a list of distinguished speakers at the morning session and the afternoon session will be devoted to a forum which will discuss a variety of subjects having to do with medical service.

AMERICAN COLLEGE OF SURGEONS

The following surgeons were recently received into fellowship in the American College of Surgeons at their convocation in Cleveland: Peachy R. Gilmer, Shreveport; William A. K. Seale, Sulphur; Gilbert C. Tomskey, New Orleans; Donald B. Williams, Lafayette.

DR. DESANCTIS SPEAKS

The exercises celebrating the completion of the new Hotel Dieu Pediatric Department included two addresses by Dr. Adolph G. DeSanctis, Director of the Department of Pediatrics of the New York Post-Graduate Medical School. Dr. Socola is chairman of the Arrangement Committee for these exercises.

A. O. A. ELECTIONS

The Tulane School of Medicine chapter of Alpha

Omega Alpha, national honorary medical fraternity, has elected 12 new members.

Seniors chosen include Hurst Bunn Hatch, Jr., Raleigh, N. C.; Erwin Hecker, Washington, D. C.; James Gentry Moore, Amarillo, Tex.; Takeshi Okano, Hawaii; Benjamin Reid Clanton, Lancaster, S. C.; George Wylie Wyatt, Amarillo, Tex.; Francis Leon Jaubert, Jr., and Aynaud Maurice Hebert, New Orleans; William Dennis Thames, Jr., Luskin, Tex.; and Thomas Everett Strain, Jr., Shreveport.

Juniors elected are Miss Flora Finch, McComb, Miss., and Francis Meredith Parks, New Orleans.

THE SCHERING AWARD

It is a pleasure to report that Mr. Stonewall B. Stickney of Tulane University School of Medicine received the third prize, a check for $200, for his contribution to the Schering Contest. It is interesting to know that in addition to the first three prizes 44 other contestants received "honorable mention" and were awarded stethoscopes.

U. S. P. H. S. NEWS

The U. S. Public Health Service will hold an examination in New Orleans March 17 at the Marine Hospital for candidates to the commissioned corps of this organization.

The Health and Hospital Division of the governor of Louisiana has been designated by the U. S. Public Health Service to administer the new Hospital Program. Hospitals and professional groups wishing to participate in the Program should direct their inquiries to Mr. Charles Mitchell, Baton Rouge, Director of the Health and Hospital Division.

TOURO INFIRMARY

The annual meeting of the Medical Staff of Touro Infirmary was held on Wednesday, January 8 at 8 p. m. The Department of Pathology presented a clinico-pathologic conference with a clinical discussion by Dr. Oscar Blitz. Dr. Guy Caldwell spoke on Correction of Discrepancies in Leg Lengths. Dr. Howard Karr discussed Diagnostic Use of Pantapaque.

GARDINER APPOINTED SUPERINTENDENT OF LAKESHORE HOSPITAL

It has been announced by the New Orleans Medical Foundation, the president of which is Dr. C. B. Odom, that Dr. W. P. Gardiner has been made superintendent of the Lakeshore Hospital. Dr. Gardiner has just completed a five year tour of duty in the Navy. He is a graduate of Tulane University School of Medicine where he obtained high scholastic honors as shown by being selected as a member of the honorary scholastic fraternity, Alpha Omega Alpha.

ROOT VISITS NEW ORLEANS

Dr. Howard F. Root, distinguished author, professor of Medicine at Harvard University, and authority on the management of Diabetic surgery at the Elliot P. Joslin Clinic in Boston, visited New Orleans as the guest of Dr. Robert A. Katz of Touro Infirmary and also of Captain E. H. Carnes and O. W. Williams of the U. S. Marine Hospital.

Dr. Root came to observe the original clinical work done here with diethyl ether in the treatment of impending gangrene. The U. S. Marine Hospital presented a program covering their experience with diethyl ether in 27 cases of peripheral disease. A demonstration of the use of atomic sodium in the measurement of circulatory improvement was also made.

MEDICAL REVIEW COURSES AT TULANE

Dr. Hiram W. Kostmayer, Director of the Division of Graduate Studies at Tulane, has announced a series of short review courses which will be held during the coming spring. Courses already have been conducted in gynecology and obstetrics, gastrointestinal diseases, and pulmonary diseases. On February 3-8, courses will be held in cardiovascular diseases; March 3-7, pediatrics; March 10-15, urinary diseases; March 17-22, nervous system diseases; March 24-29, nutritional and metabolic diseases; March 31-April 5, infectious diseases and April 7-12, neoplastic diseases. These courses will be conducted for the most part by members of the Tulane Medical School faculty, but for each course outside lecturers who are interested and prominent in the field will assist in the holding of the course.

NEWS ITEMS

Dr. Owen Wangensteen, Chief of the Department of Surgery and Professor of Surgery, The Medical School, University of Minnesota, lectured to the Fellows of the Alton Ochsner Medical Foundation on January 23, 1947 at 8:30 p. m. His subject was "The Problem of Gastric Ulcer".

In response to many inquiries from the public in reference to the use of glycols and ultraviolet radiation in control of airborne infections the National Institute of Health points out that the methods of controlling airborne infections are still within the experimental stage and no decision can be made as yet whether these methods are of value. Actually unshielded ultraviolet radiation is harmfull to the eyes and to the exposed skin of humans.

Through the cooperation of Mead Johnson & Company $34,000 in War Bonds are being offered to physician-artists (both in civilian and in military service) for best art works by physicians in war and in peace.

This contest is open to members of the American

Physicians Art Association and will be judged June 9-13, 1947 at the Atlantic City Session of the American Medical Association. For full details, write Dr. F. H. Redewill, Secretary, Flood Building, San Francisco, Cal., or Mead Johnson & Co., Evansville 21, Indiana.

Mr. Paul W. Roeder of Ciba Pharmaceutical Products, Inc., Summit, New Jersey, is very much concerned regarding the error in the slide rule they distributed to practically all of the physicians in the United States and have asked that this be called to the attention of our readers.

GROWTH OF LOUISIANA PHYSICIANS SERVICE

THE "BLUE SHIELD" PLAN

A report on the growth of Louisiana Physicians Service, Inc., is encouraging. In the first two months of active operation the company has been able to enroll 1,970 subscribers with 4,002 dependents making an aggregate of 5,972 individuals covered by the doctor's "Blue Shield" plan.

It is also encouraging to note the number of participating physicians who have voluntarily enrolled as participants in the plan. At the end of the year 517 physicians signed participating physicians agreements and as this story is going to press nearly 550 have enrolled to accept the fee schedule as contained in the policy certificate for surgical procedures as full payment for services rendered to members of the low income group, namely single individuals whose annual income is $2,000 or less and the family group whose aggregate family income is less than $3,000. Physicians participation in the plan is nearly 35% and it is hoped within a short time approximately 80% or 90% will see fit to enroll as participating members.

The company has been able to build in its two active months of enrollment an aggregate annual premium value in excess of $41,000.00. During this period it paid 13 claims submitted by the doctors and in each instance the claims were paid within a few days. This is another feature about the plan. The doctor only has to obtain the assignment of the policy certificate from the patient and then collect the amount stipulated in the Schedule of Benefits direct from the company without any red tape.

All expenses of management of the new company have been absorbed in its operation and nearly all of the preliminary expenses of organization have been absorbed in the first two months of operation.

There are still some physicians who are not familiar with the operation of the plan; however, it has been completely covered and explained in recent issues of the Journal.

At this time some might be asking. What is a prepayment surgical and obstetrical care plan? The answer is very simple. It is merely an organization that enrolls the subscribers, collects the assessment fees, and pays the benefits to the doctors for service rendered. Again these questions might be asked.

Are pre-payment surgical and obstetrical care plans in the public interest?

Are these plans good for our patients?

Are these plans good for the medical profession?

The answer to these questions is YES.

Louisiana Physicians Service, Inc., operates in the State of Louisiana and is sponsored and approved by the Louisiana State Medical Society. This plan has been granted the Seal of Acceptance of the Council on Medical Service of the American Medical Association. The plan has also been granted membership in Associated Medical Care Plans, the national organization of all pre-payment medical care plans.

From time to time you might hear the phrase "BLUE SHIELD". When a person refers to the "Blue Shield" plan he means the doctor's plan, for we bear the identification of the "Blue Shield". "Blue Shield" on a national basis means the organization of medical care plans to provide for the payment of the doctor's bill.

ASSOCIATION OF AMERICAN PHYSICIANS AND SURGEONS

On February 1, 1947, AAPS will launch its first Annual Essay Contest for Junior and Senior High School Students on the subject, "Why the Private Practice of Medicine Furnishes This Country With the Finest Medical Care."

The Association is offering $1,000 first prize; $500 second prize; and $100 third prize for the three best essays in the nation.

Essays must be limited to 1500 words and the Contest is open to Junior and Senior High School students of all schools, except sons and daughters of physicians.

Your Parish Medical Society is urged to sponsor a local contest and offer local prizes for the three best essays in your Parish. In the event your State Medical Society sponsors a state contest, the three winning essays in your Parish should be forwarded to State headquarters to compete for state prizes, with the three best essays in the State being sent on to the AAPS National Headquarters for participation in the National Contest. However, if your State Society does not sponsor a State Contest, send the three winning essays from your Parish directly to the Headquarters of the Association of American Physicians and Surgeons at 11 South LaSalle, Chicago 3, Illinois, for entry in the National Contest which ends March 30.

Medical leaders throughout the country believe that the AAPS Annual Essay Contest will constitute the most important contribution ever made to good public relations of organized medicine. Participation in the Contest will require high school boys and girls to do research to become familiar with the reasons why the private practice of medicine provides this country with the finest medical care in the world. The contest affords medicine the opportunity of having its true and praiseworthy story reach the youngsters of all levels of population, and through them, their parents and teachers. Further, the Contest provides the means for medicine to dig deep in the grass roots of public opinion with the factual narrative of its accomplishments in the public interest.

FELLOWSHIPS FOR PHYSICIANS AND ENGINEERS

Announcement is made by Surgeon General Thomas Parran of the U. S. Public Health Service that applications for Fellowships in post-graduate public health training for physicians and engineers for the school year beginning in the fall of 1947 will be received at any time prior to May 1, 1947.

The Fellowships are made possible by a grant of $228,400 from the National Foundation for Infantile Paralysis through funds contributed to its March of Dimes. Fifty-three students were awarded Fellowships for the school year beginning in September 1946.

The specific purpose of the Fellowships is to aid in the recruitment of trained health officers, directors of special services, and engineers to help fill hundreds of vacancies existing in State and local health departments throughout the country. The Fellowships are intended for newcomers to the public health field, and are not open to employees of State and local health departments, for whom Federal grant-in-aid funds are already available to the States.

Applicants for Fellowships may secure further details by writing to the Surgeon General, U. S. Public Health Service, 19th and Constitution Avenue, N. W., Washington 25, D. C., Attention Public Health Training.

U. S. P. H. S. EXAMINATIONS

Competitive examinations will be held early in 1947 for appointment to the Regular Corps of the United States Public Health Service, according to a recent announcement by Dr. Thomas Parran, Surgeon General. Seventy-five vacancies exist in grades of Assistant and Senior Assistant Scientist.

Written examinations, covering each candidate's particular field of science, as well as related fields, will be held April 14 and 15 at places mutually convenient to the applicant and the Service. Oral examinations will be held during the period February 13-April 9 in thirty cities, strategically located throughout the United States; New Orleans, February 14.

Application forms and additional information may be obtained by writing the Surgeon General, United States Public Health Service, Washington 25, D. C.

PHYSICIANS WANTED

A physician qualified to do general practice, including obstetrics, is needed by the Myles Salt Company at Weeks, Louisiana. Equipped office furnished.

For further information contact Mr. Robert H. Polack, Vice-President, Post Office Box 329, New Orleans 9.

The office of the State Society has received information that there is need for a physician in Buras, Louisiana. Office space consisting of three rooms, including a small laboratory equipped with gas jet, work table, desk and running water is available.

If interested contact Mr. Peter C. LaDart, LaDart's Pharmacy, Buras, Louisiana. Telephone 2431.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending December 7 there were 130 deaths in the city of New Orleans as contrasted with 149 the previous week and 160 for the 3-year-average corresponding week. These deaths were divided as follows: white 93, non-white 37; 12 of the deaths occurred in children under one year of age. For the week which came to a close on December 14, there were 14 more deaths reported in the city of New Orleans than in the previous week. These were apportioned 91 white, 53 colored and 17 infants, most of whom were negro children. There were approximately the same number of deaths in the city for the week ending December 21 as the previous week, 146 versus 144. Of the total number of deaths, 99 occurred in the white population, 47 in the negro, with only seven infant deaths in this particular week. For some reason or another there was a sharp increase in the number of people dying in the city for the week ending December 28 as contrasted with the previous week. However, the number of deaths was still considerably smaller than the 3-year average; 102 of the white population died in the week closing December 28, 70 negroes expired and there were 11 infant deaths.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending December 14 showed the following reportable diseases in numbers greater than ten; 25 cases of pulmonary tuberculosis, 20 of hookworm infestation and 13 of cancer. This is really quite a remarkable health record. There were three cases of poliomyelitis reported from three different parishes and two cases of typhus fever. For the week which ended December 21, pulmonary tuberculosis led all reportable diseases with 61 cases followed by 21 of cancer, 27 of unclassified pneumonia, 14 of hookworm infestation and 10 of lobar pneumonia. The few cases of poliomyelitis, three, as were reported this week again, had their origin in three different parishes of the state. Probably the most unusual weekly morbidity report that has come to the Journal Office in many years was that of December 28 when the 43 cases of influenza which were listed represented the only reportable disease that occurred in frequency greater than eight. It may be that the Christmas season had a salubrious effect on the health of the Louisiana people or it may be that the Christmas cheer postponed the reporting of reportable cases.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending November 30, 1946

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	39	556	595
Gonorrhea	1239	15756	16995
Granuloma Inguinale	28	252	280
Lymphopathia Venereum	2	97	99
Syphilis	1290	16960	18250

J. CYRIL EBY

1884-1946

Dr. Eby died suddenly of coronary occlusion in New Orleans while doing Christmas shopping. Dr. Eby was a graduate of the Maryland College of Pharmacy of the University of Maryland, 1916. He served during the first World War and then moved to Plaquemine in 1920. He soon became connected with the State Health Department and at the time of his death was the Iberville Parish Health Officer, serving also during the last war on account of the shortage of health officers in Ascension Parish.

WOMAN'S AUXILIARY

HIGHLIGHTS AND PROGRESS NOTES ON THE NATIONAL CONFERENCE OF PRESIDENTS AND PRESIDENTS-ELECT

The first joint meeting of the Board of Trustees of American Medical Association and the Board of the Woman's Auxiliary has taken place! It means that for the first time in the twenty-five years of its existence the Woman's Auxiliary has really come into its own and been recognized as a powerful asset to the A. M. A. A definite program of action for the Auxiliary will be formulated. Time and again, throughout most of the speeches by the doctors who spoke to the Conference it was brought out that the greatest challenge to doctors' wives was in the field of public relations. The task of educating the public to the aims and accomplishments of the medical profession, and teaching the desirability of the wide-spread use of voluntary medical insurance is one of the greatest services that the women of the Auxiliary can perform. But two points were stressed in this connection: *First*, that the best public relations were those which were the least obvious. Don't label open meetings as "Public Relations teas". Public relations is "just making friends",—and exhibiting a real interest in local problems and community welfare is the best way to cement finer Public Relations. *Secondly*, we must alter our attitude. We must *not* say the Voluntary Prepayment Plans are "our only weapon for fighting socialized medicine". Voluntary Prepayment Plans were designed, not to defeat the Wagner-Murray-Dingle bill, but to give *better* medical care to *more* people. The quality of medicine is above reproach; only the distribution is lacking.

Most state Auxiliaries now have study groups in which auxiliary members are learning about their local Voluntary Prepayment Plans, so that they can talk intelligently to lay persons.

Most states now hold annual Schools of Instruction for incoming officers and chairmen to familiarize them with their new duties. Louisiana plans to hold one at the Post Convention Board meeting.

Many states now have a mid-year conference of Presidents and Presidents-Elect patterned on Nationals, with round-table discussions. Louisiana approaches this plan in the mid-year Executive Board meeting.

Louisiana is the first state to hold state-wide open public relations meetings with a speaker to expose the dangers inherent in socialized medicine such as that proposed in the Wagner-Murray-Dingle bill. Mrs. Rex Fortenberry, a lawyer from Beaumont, Texas, has completed a tour of the entire state, speaking in all eight districts and contacting between 3,000 and 3,500 people.

Louisiana is the first state to ask the cooperation of the Auxiliary in aiding in the Survey of the American Academy of Pediatrics which is now being conducted on a national scale. And from reports of our local state chairman, Dr. C. H. Webb, the results of the Auxiliary's efforts in Louisiana are very gratifying. Louisiana's returns on the survey of general practitioners are 15.6 per cent higher than the pilot surveys!

Louisiana is now one of three states to publish an outline of its annual program. The other two are California and New York. Numerous requests for copies of the outline have been received from other states.

Several states have found that a membership file that lists all Auxiliary offices and activities of a member, as well as other club affiliations, to be an invaluable asset to the state officers and nominating committee in choosing officer material. The national treasurer also asked that state treasurers be retained for more than one year, if possible.

It was also suggested that Public Relations chairmen urge that an up-to-date public relations file be kept on all local civic leaders of lay groups for facilitating speedy contacts with the proper persons to organize open meetings, health education programs, etc.

Debating kits are prepared and distributed to high schools, colleges and public libraries by many state auxiliaries. The Committee of Medical Services is prepared to furnish reference materials for these kits on Voluntary Prepayment Plans vs. Compulsory Insurance.

The Bureau of Health Education has two new members, Dr. Smiley of Cornell and Dr. Fred Hime, Ph. D. of Wisconsin, whose services as public speakers are available at A. M. A. expense to all states. They are expert consultants in the educational field.

Dr. W. W. Bauer, Director of Bureau of Health Education, also urged the wider use of electrical radio transcripts. There are fourteen series of platters on a variety of medical subjects now available together with the posters, press releases and necessary publicity, to furnish a fifteen minute program once a week for thirteen weeks, free of charge. Since program directors of local radio stations are required by law to make a report every three years on the amount of time *given* to public service projects they are usually more than glad to cooperate with any worthwhile organization.

Dr. Morris Fishbein reviewed the extensive plans being made for the celebration of the A. M. A.'s 100th anniversary at the Centennial Convention to be held in Atlantic City next June. A varied and imposing array of personalities and talent is being assembled for this meeting including such noted figures as Dr. Conant of Harvard, Kettering of General Electric, Monsignor Fulton Sheenan, Frank Howe Richardson, Zimbalist, Fred Waring and Guy Lombardo. There will be a March of Time recording the evolution and progress in the realm of medical science; the Postmaster General will probably endorse the issue of a special stamp commemorating the event; a history of the A. M. A. and the Medical Auxiliary is being prepared; and over 25,000 are expected to attend the largest convention ever held. I hope to see you in Atlantic City. (Further details will be printed in the next issue of the Journal.)

BOOK REVIEWS

Skin Diseases; Nutrition and Metabolism: By Erich Urbach, M.D., F.A.C.A., New York, Grune & Stratton, 1946. Illus. 634 p. Price, $10.00.

This new book contains a description of the metabolism and biochemistry of the skin, detailing the water, mineral, carbohydrates, lipid and protein metabolism of the skin with charts, reference to studies and experiments which clarify many of the modern theories of the skin's action. To the student who wants in a small chapter a concise knowledge of skin metabolism, a reader could not find the same amount of material in a whole volume.

The chapter on the Influence on Diet on Metabolism of the Skin, probably over-estimates the effect of diet on many diseases. The European idea of the beneficial effect of the Gerson and Sauerbruch-Hermannsdorfer diets is fully lauded but should be of great interest to younger students who have not acquainted themselves with the literature of the last generation. In part II of this volume the author discusses nutritional causes of dermatoses, listing those due to malnutrition, vitamin deficiencies, food allergies and alimentary infections and alimentary intoxications.

Allergy is discussed, described and many suggestions are given for the treatment of many skin diseases considered by the author to be of nutritional origin. The student of skin diseases, internal medicine, allergy and pediatrics should find this book a store house on the chemistry, physiology and metabolism of nutrition of the skin in health and disease.

M. T. VAN STUDDIFORD, M. D.

Synopsis of Genitourinary Diseases: By A. J. Dodson, M. D., F. A. C. S. 4th ed. St. Louis, C. V. Moshy Co., 1945. Pp. 313, illus. Price, $3.50.

This excellent book was designed to present a synopsis of genitourinary diseases, which could be easily assimilated by medical students, and to serve as a handy reference for the general practitioner. Etiology, pathology and diagnosis are stressed. Treatments are recommended, but the technics of procedures are necessarily omitted in this small edition.

The fourth edition includes a more comprehensive discussion of the uses and toxicity of the sulfonamide drugs, the use of penicillin and its application, a listing of bacteria which may be responsible for urinary tract infections, and the value of castration and endocrine therapy in the treatment of carcinoma of the prostate. The use of streptomycin is not included.

This book has admirably fulfilled the aims of its author, and is recommended to medical students, general practitioners and all physicians interested in urology.

JOHN G. MENVILLE, M. D.

Women in Industry: Their Health and Efficiency: By Anna M. Baetjer, Sc. D. Philadelphia and London: W. B. Saunders Co., 1946. 122 tales and 4 appendices, pp. 344. Price $4.00.

This study, begun during the war and carried out in the Army Industrial Hygiene Laboratory, is an exacting analysis of an exhaustive review of the literature on the question of utilization of womanpower in industry. Dr. Baetjer, throughout the book, made no attempt to justify the employment of women in any type of industrial work, either new or old to them, but did devote herself toward (1) making available any information that might be of value in proper placement of women when their service might be required in industry, (2) making an evaluation of any occupational effects on the physiologic processes in women, and (3) offering practical recommendations for industry in dealing with these problems.

The text is presented under six discussions of study: the ability of women to work, sickness absenteeism among women employees, accidental injuries to working women, occupational diseases, gynecologic and obstetrical problems associated with the employment of women, and mortality and fertility of women in relation to occupation. One hundred and twenty-two carefully planned tables are used. Four extensive appendices, an exhaustive 23-page bibliography, and a satisfactory index round out the book.

This volume will be of very definite interest as a handbook for industrial physicians and personnel managers as well as for members of industrial relations groups.

Ralph H. Heeren, M. D.

The Venereal Diseases: By James Marshall, M. B., B. S., M. R. C. S., L. R. C. London, Macmillan & Co. Ltd., 1944. Pp. 348.

The author has succeeded in writing a general review of the diagnosis and treatment of venereal diseases, which should prove very helpful to general practitioners and students.

The book is divided into four parts, namely, gonorrhea, part I; syphilis, part II; other venereal and allied diseases, part III, and technic, part IV.

In part I, a short practical description of the anatomy of the genital organs of both sexes, the symptoms and signs of the disease, diagnostic procedures and the treatment of gonorrhea and its complications in the male and female are presented. Aspects of gonorrhea in both sexes, vulvovaginitis in children, urethral stricture and sulfonamides are discussed. Unfortunately, penicillin was in the experimental stage and all of the sulfonamides were not available in sufficient quantities when the book was written. This explains the space devoted to sulfapyridine in the treatment of gonorrhea.

In part II, syphilis, its complications and treatment, are covered in a well organized manner. The stages of the disease and its complications are extremely well illustrated and are invaluale to the student of this disease. The discussion of the drugs and the suggested courses of treatment are well outlined, efficient and comprehensible. Penicillin unfortunately is not included in the proposed treatments.

In part III, other venereal and allied diseases are discussed and a short practical synopsis of each is given.

In part IV, technic and practical instructions are covered and are valuable aids to all not versed in the common urological procedures.

The book is comprehensive, extremely well illustrated, well organized and practical. It should be a valuable guide to the general practitioner and student. Experience with penicillin and the commoner sulfonamides are not included.

John G. Menville, M. D.

New and Nonofficial Remedies, 1946, containing descriptions of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association on Jan. 1, 1946. Chicago, American Medical Association, 1946. Pp. 770. Cloth. Price, postpaid, $1.50.

New and Nonofficial Remedies is the book in which are listed and described the medicinal preparations which the Council on Pharmacy and Chemistry has found acceptable, under its rules, for the use of physicians. To have a product accepted, the manufacturer must declare its composition, give adequate proof of its therapeutic value and market it with claims which have been found valid by the Council. The present volume represents a cumulative epitome of the Council's work since its foundation in 1905.

Accepted preparations are grouped in twenty-four classifications ranging from allergenic preparations to vitamins. Ordinarily, an inclusive general article precedes the description of the various products. The monograph for the products set forth the actions, uses and dosage and usually a set of tests and standards. As its name implies, the book is intended to describe nonofficial preparations, that is preparations which are not included in such official publications as the Pharmacopeia and the National Formulary. However, some official articles are listed and described, these being in general those for which the Council feels the practicing physician needs concise and authoritative information. In the preface of the present volume, the Council lists some thirty-five official drugs ranging from acetylsalicylic acid to strophanthin, which the Council feels it no longer necessary to consider for inclusion in the book. However, in most cases, a brief monograph on actions, uses and dosage gives information useful to the physician and for the control and advertising of marketed preparations.

Examination of the volume reveals that there have been no extensive or radical revisions of the general articles representing the twenty-four chapter heads under which preparations are classified. A few revisions of separate monographs may be mentioned: under chaulmoogra derivatives, the recommended use of chaulmoogra oil is limited to sarcoidosis; the dosage statement for quinacrine hydrochloride has been notably expanded to reflect the war-time experience with the drug. The radically revised monograph on amphetamine is in harmony with the recent Council report on the use of this drug. Minor revisions of the chapter on contraceptives are noted, and one marks the appearance of many additional products. The monograph on the vitamin B complex now mentions synthetic folic acid, recently made available for investigational use; but no accepted preparations are listed.

There appear to be no spectacularly new accepted preparations. Perhaps the most noteworthy is the casein hydrolysate, amigen, acceptance of which will no doubt be followed by that of many more preparations representing the field of amino acid therapy.

Diabetes—A Concise Presentation: By Henry J. John, M. A., M. D., F. A. C. P. St. Louis, The C. V. Mosby Company, 1946. Pp. 300. Price $3.25.

This is a rather unusual type of treatise on diabetes mellitus and seems to consist of a series of essays on various features of diabetes, not a concerted discourse on the disease itself. Thus there is no presentation at all of the relationships of diabetes and peripheral vascular disease, ocular disorders, various types of infectious diseases, of cardiac disease or any detailed discussion of the forms of insulin available today.

Dr. John is firmly convinced that hyperglycemia must not be neglected ("A continued hyperglycemia destroys a pancreas in time") one day after the diagnosis has been established, although many authorities will not agree with his contention. He believes that examination of the urine four times a day, as is so commonly practiced, is a very poor means of controlling the severity of the diabetes; he insists on a daily 24 hour estimation of urinary output of glucose and determination of the blood sugar levels three or four times a day while the patient is first under observation.

One chapter entitled "Some 'Do's' And 'Don'ts' " is given over to some detailed bits of advice reflecting the author's philosophic views on the treatment of patients in general and diabetics in particular. It comprises a range of topics from the psychologic approach to the patient to suggestions for performing venipuncture.

SYDNEY JACOBS, M. D.

The Modern Treatment of Diabetes Mellitus: By William S. Collens, B. S., M. D., and Louis C. Boas, A. B., M. D. Springfield, Illinois, Charles C. Thomas, 1946. Pp. 514. Price $8.50.

This is a good treatise on diabetes mellitus and fulfills its purpose of acting as "a practical comprehensive guide for the general practitioners who treat diabetic patients". In line with the current tendency to simplify the treatment of diabetes mellitus, the tone of the book is almost dogmatic at times.

The historical introduction is unusually well-written and informative. The conventional presentation of allotting a chapter to each of the important considerations of diabetes is carried to the logical extreme of a therapeutic classification. Thus according to the severity of the illness, certain differing modes of therapy are given. Although this makes for some repetition, the result is good. In all chapters, the attempt is made to present to the clinician a maximum of applicable data and a minimum of "theoretical" background.

Several features deserve comment. The chapter on vascular diseases is exceptionally good; the use of illustrations in color materially improves the presentation. In view of the long interest of both authors in peripheral vascular disease, this is not remarkable. The direct and pointed suggestions for the solution of problems of vascular disease (either peripherally or of the coronary system) is most valuable. For an unexplained reason, the relationship of high fat diets to the incitement and aggravation of peripheral vascular disease is not included.

The use of "clock" diagrams to point out the effects of modified and of unmodified insulins during a 24 hour period is helpful in deciding which form of insulin to use under a given set of circumstances. The liberal use of photographs, charts and sketches is additionally illustrative. Important points are placed in italics or in bold face type, thus compelling attention.

The chapter on diabetes of the pregnant woman is rather scantily presented, and the hormonal approach to prevention and therapy of the toxemias is not critically stated. Sterility of the diabetic, the use of estrogenic substances to modify the diabetes of middle-aged females, the occurrence of hemochromatosis and the co-existence of tuberculosis and diabetes are not mentioned in the book.

Despite these deficiencies, it is to be recommended to anyone wishing a practical method of handling the more frequent diabetic problem.

SYDNEY JACOBS, M. D.

Progress in Neurology and Psychiatry: Ed. by E. A. Spiegel. New York, Grune & Stratton, 1946. Pp. 708. Price, $8.00.

Increased interest in neuropsychiatric problems has been responsible for many valuable treatises on these subjects. This volume has the distinction of being the first of a series of annual reviews which are not intended to replace existing abstract services but they will attempt to supplement them by providing critical surveys of the basic theoretical foundations as well as of various clinical aspects of neurology and psychiatry.

Edited by Doctor E. A. Spiegel, a distinguished scientist and investigator and a prodigious worker in the field of neuropsychiatry, this work includes some thirty-five contributions by an equal number of eminent specialists. Each chapter adequately surveys its special subject. All sections are splendidly done and those chapters on Psychoanalysis, Group Psychotherapy and the parts dealing with tics and management of speech disorders, are excellent.

The book as a whole is an up-to-the-present refresher course which brings one all the important advances. It is a comprehensive readable review which might be added to every physician's library and used with profit.

C. P. May, M. D.

Harvey Cushing, a Biography: By John F. Fulton, Springfield, Ill., Charles C. Thomas, 1946. Por. pp. 754. Illus. Price, $5.00.

The story of Harvey Cushing (1869-1939) by his literary executor, the eminent Dr. John F. Fulton, may not appeal to those who have acquired a taste for fictionized biography. Other biographers and students of medical history, and of character, undoubtedly will commend the meticulous method by which are recorded the life events of a master surgeon and man of letters. Considerable time will be required for careful reading of the more than 750 pages of text, the greater portion of which is composed in eight and nine point type, but there is rich reward to be gained in following the mental and spiritual development of the extraordinary individual whose portrait is honestly and understandingly presented.

Anticipating that an account of his life might be "of interest or help to medical students," Cushing provided funds for compiling this volume from which royalties will be paid to the Medical Historical Library of Yale University. Dr. Fulton accepted his difficult assignment in much the same spirit that inspired Cushing to write his imperishable *Life of Sir William Osler*, the Pulitzer prize-biography of 1925. With selective judgment he has chosen from an abundance of source material concerning the external life of his friend who became the leading neurological surgeon in the world. Interwoven are many illuminating incidents in the lives of heroic medical men — Osler, Halsted, Welch, Klebs, Kelly, Finney, Sherrington, John Homans and others with whom he was associated. Of no less interest are the accounts of his associations with the great medical institutions of Yale, Harvard, John Hopkins, Massachusetts General, and Peter Bent Brigham hospitals.

Throughout the voluminous correspondence that is included are intimate glimpses of the inner man which enlarge our understanding of his complicated character. Diaries from student days, descriptions of travels illustrated by his facile pencil, cuttings from files and scrapbooks, letters to his family which had been carefully preserved by his father — in these does one discover Cushing's great love of nature, his pride in his illustrious forebears, his strong family ties and devotion to his physician-brother "Ned," his interest in his patients and their families, his concern with needy students, wartime experiences, occasional conflicts, his professional attainments, honors and recognition — one can only suggest the wealth of material that is revealed in this sharp and clear delineation of a great and a useful life. Throughout each chapter the reader is ever conscious of the motivating force that enabled Cushing to succeed in varied fields of endeavor, of his driving ambition to attain perfection and his almost fanatical passion for work to which his life was devoted even through periods of physical weakness.

In our opinion, the book succeeds in what has been termed the "most delicate and humane of all forms of writing" and will serve as an inspiring example for students, holding interest for all who are concerned with the problems of medical education and of practice. Dr. Fulton has made an enduring contribution in his historic volume from which we quote the closing words: " 'By valor and divine aid,' he (Cushing) had more than fulfilled the promise and ambition of his youth: scientist, pathfinder, artist, writer, and bibliophile — yes, and above all, 'a good doctor'."

Dean H. Echols, M. D.

PUBLICATIONS RECEIVED

Comstock Publishing Co., Inc., Ithaca, N. Y.: Insect Microbiology, by Edward A. Steinhaus, Assistant Professor of Bacteriology and Assistant Insect Pathologist.

Duke University Press, Durham, N. C.: The Compleat Pediatrician, by Wilburt C. Davison, M. A., D. Sc., M. D.

Harcourt, Brace and Company, New York: Animal Farm, by George Orwell.

The John Hopkins Press, Baltimore: Henry Sewall, Physiologist and Physician, by Gerald B. Webb and Desmond Powell.

Lea & Febiger, Philadelphia: The Essentials of Obstetrics and Gynecology, by William Albert Scott, B. A., M. B., F. R. C. S., F. R. C. O. G., and H. Brookfield Van Wyck, B. A., M. B., F. R. C. S., F. R. C. O. G.

W. B. Saunders Company, Philadelphia: An Integrated Practice of Medicine (Index and 4 Volumes), by Harold Thomas Hyman, M. D.

Washington Institute of Medicine, Washington, D. C.: Quarterly Review of Obstetrics and Gynecology, Volumes 1 (1943), 2 (1944), and 3 (1945).

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and
Surgical Journal

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HEART DISEASE IN CHARITY HOSPITAL OF LOUISIANA IN NEW ORLEANS*

A SURVEY OF 8,313 ROUTINE AUTOPSIES

JOE E. HOLOUBEK, M. D.†
ALICE BAKER HOLOUBEK, M. D.†
SHREVEPORT
AND
DeWITT T. BAKER, M. D.
NEW ORLEANS

A statistical analysis was made of all of the autopsies in which the deaths were attributed to heart disease at the Charity Hospital of Louisiana at New Orleans between the years 1935 to 1940 inclusive. The criteria of classification of the etiological causes of heart disease of the American Heart Association were used.††

During this period there were 19,623 deaths in the hospital of which 9,178 or 46.3 per cent were in the white race and 10,445 or 53.8 per cent were in the negro (table 1). There were 8,313 autopsies performed during this time or a percentage of 42.3 of the total deaths. Autopsies were obtained in one-third of the white deaths and 50.3 per cent of the negro deaths. Of the total number of autopsies during this period, 5,252 or 63.2 per cent were in the negro race. There were 4,957 males (59.6 per cent) of both races in the series of autopsies.

There were 1,045 autopsies (12.6 per cent of the total number) in which some type of heart disease was the primary cause of death. These varied in age from stillborn to 98 years. The negro race made up 63.6 per cent of this total number. This is almost identical to the percentage (63.2) of the negro race in the total number of autopsies.

The male sex comprised 67 per cent of the total deaths due to heart disease and 59.6 per cent of the total number of autopsies.

TABLE 1

Total Deaths, Autopsies and Cardiac Deaths at Charity Hospital at New Orleans (1935-1940)

	White Race				Negro Race				Grand
	Male	Female	Total	Per cent	Male	Female	Total	Per cent	Total
Total deaths	5729	3449	9178	46.2	6031	4414	10445	53.8	19,623
Total autopsies	1975	1086	3061	36.8	2982	2270	5252	63.2	8,313
Total Cardiac deaths	281	99	380	37.4	423	242	665	63.6	1,045

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*Read before the sixty-sixth annual meeting of the Louisiana State Medical Society, Alexandria, May 6-8, 1946.

†Formerly of the Department of Medicine of the Louisiana State University School of Medicine and the Charity Hospital at New Orleans.

††The members of the Pathology Department of the Hospital and of the Louisiana State University School of Medicine were very cooperative, and assisted in the study of records which proved difficult to interpret.

TABLE 2

Incidence of Death due to Heart Disease

Etiology	White Race M.	F.	Total	Per cent	Negro Race M.	F.	Total	Cent	Grand Total	Per Cent Total
Hypertensive	101	28	129	59.4	185	109	294	69.5	423	40.5
Arteriosclerotic	96	33	127	30.5	54	34	88	40.6	217	20.7
Syphilitic	23	4	27	14.0	125	38	163	86.0	190	18.2
Rheumatic	33	17	50	42.7	34	33	67	57.3	117	11.1
Congenital	4	5	9	39.2	2	12	14	60.8	23	2.2
Toxic	8	5	13	59.1	5	4	9	40.9	22	2.1
Bacterial	9	4	13	59.1	6	3	9	40.9	22	2.1
Anemia	4	0	4	57.1	3	0	3	42.9	7	0.7
Avitaminosis	2	0	2	40.0	1	2	3	60.0	5	0.5
Combined	1	3	4	80.0	1	0	1	20.0	5	0.5
Hyperthyroidism	0	0	0	0	3	1	4	100	4	0.4
Pulmonary disease	0	0	0	0	2	2	4	100	4	0.4
Other	0	0	0	0	2	1	3	100	3	0.3
Unknown	0	0	0	0	0	2	2	100	2	0.2
Trauma	0	0	0	0	0	1	1	100	1	0.1
Total	281	99	380	36.4	423	242	665	63.6	1045	

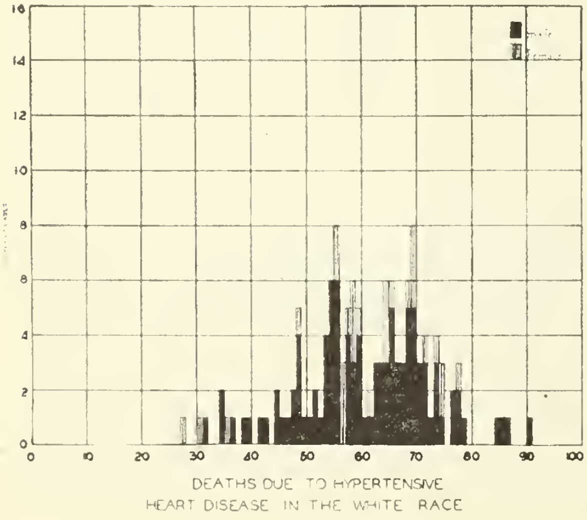
Fifteen etiologic types of heart disease were found in this survey (table 2). As will be seen, the four most frequent types of heart disease: hypertensive, arteriosclerotic, syphilitic, and rheumatic made up 90.5 per cent of the total cardiac deaths. In this group there were 612 or 64.6 per cent in the negro race and 335 or 35.4 per cent in the white race. This compares with 63.2 per cent of negroes in the total autopsy series. These will now be discussed more in detail.

HYPERTENSIVE HEART DISEASE

There was a total of 423 deaths (40.5 per cent) due to this type of heart disease, 129 in the white race and 294 in the negro race.

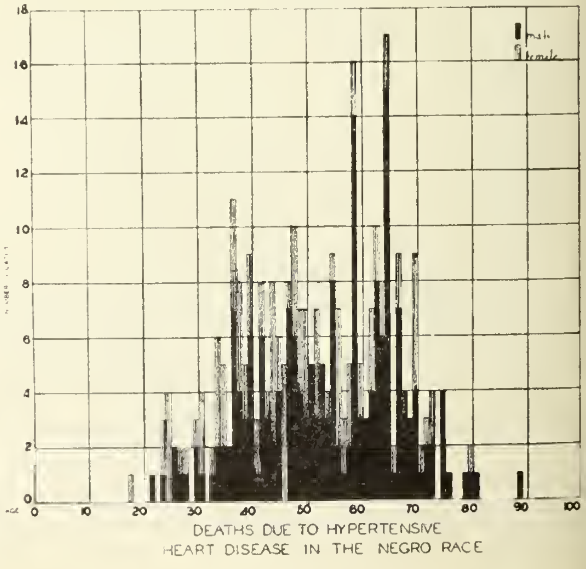
White race: There were 129 or 30.5 per cent deaths in the white race (figure 1). Of this group the males made up 70.6 per cent or a ratio of 2.33 to 1. This is compared to the ratio of males to females in the routine white autopsies of 1.72 to 1. The peak of incidence of the deaths occurred at the age of 55 years and with a gradual rise starting at the age of 45 and a sharp drop after 75 years.

Negro race: A total of 294 deaths or 69.5 per cent occurred in the negro race (fig. 2). This compares favorably with the 63.2



DEATHS DUE TO HYPERTENSIVE HEART DISEASE IN THE WHITE RACE

Fig. 1



DEATHS DUE TO HYPERTENSIVE HEART DISEASE IN THE NEGRO RACE

Fig. 2

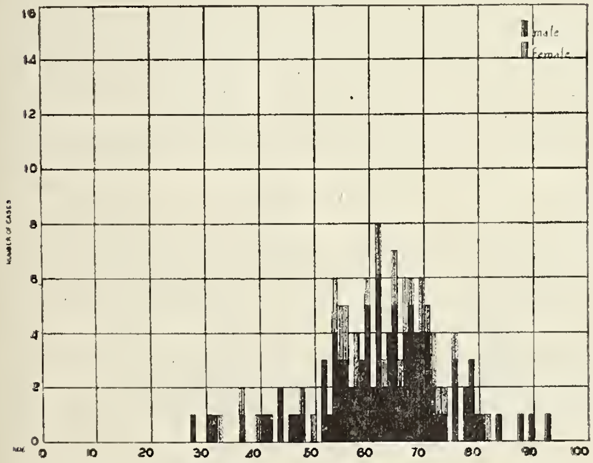
per cent of the total autopsies in the negro race. The males again predominated, there being 185 or 63 per cent, a ratio of 1.58 to 1 as compared to a ratio of negro males to females in total autopsies at 1.31 to 1. There is a sharp rise in incidence at the age of 30 years, occurring about 15 years earlier than in the white race. The peak is reached at 70 years.

The most commonly associated cardiac complication in this entire group was arteriosclerosis occurring in 143 or 33.6 per cent of the cases. Rheumatic heart disease was found in 4 per cent or 17 cases, and syphilitic heart disease in 1.8 per cent or eight cases.

ARTERIOSCLEROTIC HEART DISEASE

Two hundred and seventeen deaths (20.7 per cent) were due to arteriosclerotic heart disease, 129 (59.4 per cent) in the white race and 88 (40.6 per cent) in the negro race.

White race: There were 129 deaths (59.4 per cent) in the white race which were placed in this group (fig. 3). Included in this were six cases of dissecting aneurysm; five males aged 28, 52, 54, 55, and 62, and one female aged 58.

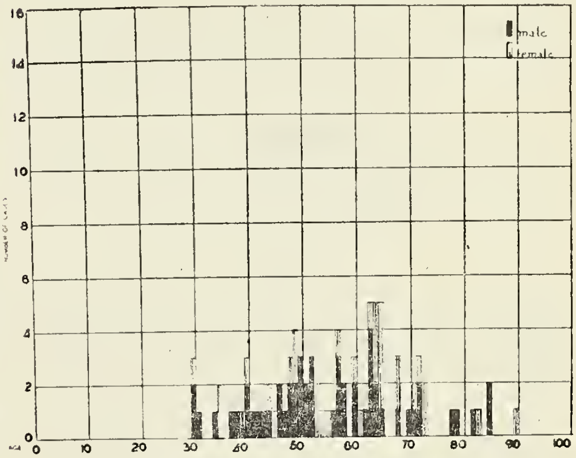


DEATHS DUE TO ARTERIOSCLEROTIC
HEART DISEASE IN THE WHITE RACE

Fig. 3

There was a total of 73 deaths due to myocardial infarction in this race, with the males predominating in a ratio of 3 to 1. In this group the males showed a sharp rise in incidence at the age of 50. This remained constant until the age of 65. Of the remaining 36 male and 14 female patients no recent evidence of infarction was found.

Negro race: There were 88 cases in this group, 54 males and 34 females (fig. 4). Of the five cases of dissecting aneurysm, four were males and one female. Myocardial infarction was found in a total of 44, 27 males and 17 females or a ratio of 3 to 2. In the remainder, 23 males and 16 females, no evidence of recent infarction was found.



DEATHS DUE TO ARTERIOSCLEROTIC
HEART DISEASE IN THE NEGRO RACE

Fig. 4

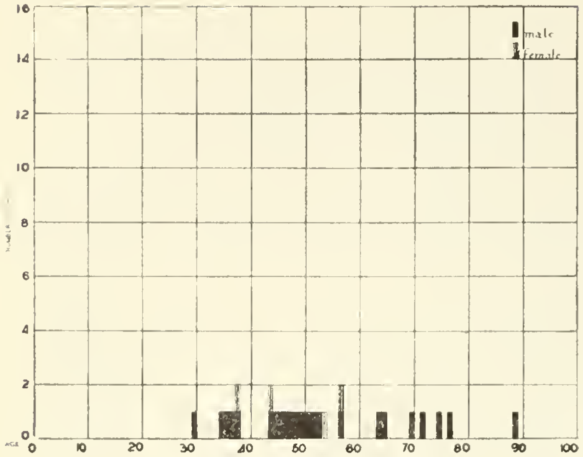
The complicating conditions in this group were; hypertension, 47 cases (21.6 per cent); rheumatic heart disease, 10 cases; and syphilitic heart disease, three cases.

Of particular interest are the infarctions occurring below the age of 40 years. There were six negro males, two negro females, two white males and two white females.

SYPHILITIC HEART DISEASE

A total of 190 (18.2 per cent) of the total deaths were due to syphilitic heart disease. Included in this group are 77 cases in which syphilitic aortic aneurysms were the cause of death.

White race: There were 23 white males and four white females in this group (fig. 5) or a ratio of almost 6 to 1. Of this

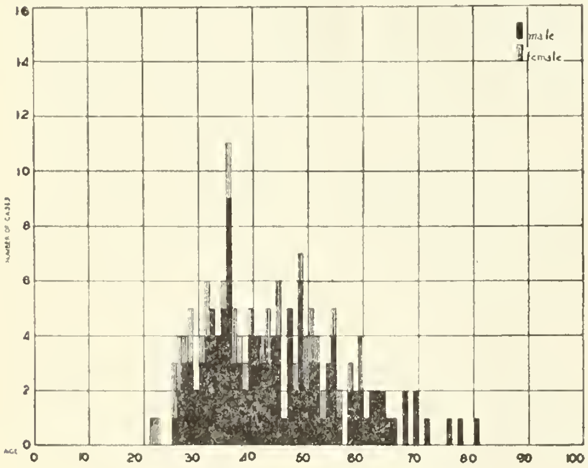


DEATHS DUE TO SYPHILITIC HEART DISEASE IN THE WHITE RACE

Fig. 5

number eight males and one female had large aortic aneurysms while the remainder had luetic valvulitis.

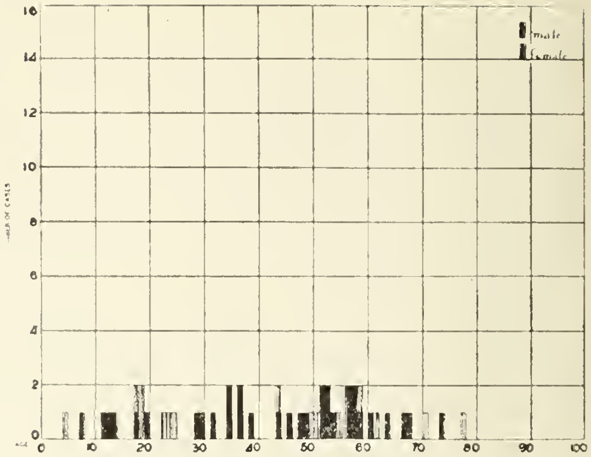
Negro race: There were 125 male and 38 female deaths occurring in the negro race, making a ratio of 3.3 to 1, males to females. There were 68 aneurysms with a ratio of five males to two females (fig. 6).



DEATHS DUE TO SYPHILITIC HEART DISEASE IN THE NEGRO RACE

Fig. 6

Complicating cardiac conditions in the entire group of syphilitic heart disease were: rheumatic, 14 cases; arteriosclerotic, 13 cases; bacterial endocarditis of the involved aortic valve, six cases; and hypertension, five cases.



DEATHS DUE TO RHEUMATIC HEART DISEASE IN THE WHITE RACE

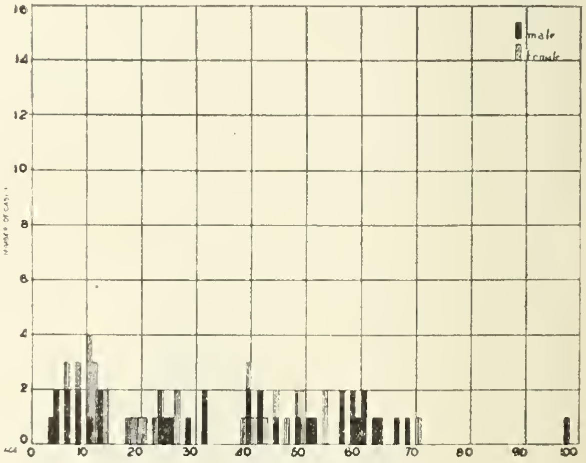
Fig. 7

RHEUMATIC HEART DISEASE

Eleven and one-tenth per cent or 117 died primarily of rheumatic heart disease.

White race: The white race made up 50 or 42.7 per cent of this group, there being 33 males and 17 females or a ratio of 2 to 1 (fig. 7).

Negro race: In the negro race the ratio was 1 to 1 with 34 males and 33 females (fig. 8). As will be seen in the graph there is a sharp rise in incidence of negro deaths in the adolescent period. A second rise occurs in the middle age group, at the age of 45 in the negro and at 60 in the white race.



DEATHS DUE TO RHEUMATIC HEART DISEASE IN THE NEGRO RACE

Fig. 8

There were 16 cases of subacute bacterial endocarditis, 11 of arteriosclerosis, five of acute bacterial endocarditis and five of hypertension in this entire group.

CONGENITAL

There were 23 deaths or 2. 2 per cent due to congenital heart lesions. These were subdivided into four white males, five white females, two negro males, and 12 negro females. The majority of deaths in this group were in infancy and the oldest was 16 years of age. This may explain the occurrence of only one case of subacute bacterial endocarditis.

TOXIC

There were 22 deaths (2.1 per cent) due to toxic heart disease, eight white males, five white females, five negro males, and four negro females. Of these 12 were due to diphtheria, four to typhoid fever, two to septicemia, two to pertussis, one to bronchopneumonia, and one to overdosage of digitalis.

BACTERIAL INFECTION

There were 14 deaths due to subacute bacterial endocarditis, six to acute bacterial endocarditis, one to acute postpneumonic suppurative myocarditis and pericarditis, and one to tuberculous pericarditis causing cardiac tamponade. There were 13 white patients, nine males and four females; and nine negro patients, six males and three females. None of these were found to show any evidence of pre-existing heart disease of any type.

ANEMIA

There were four white and three negro deaths due primarily to anemia, a total of seven or 0.7 per cent. The negro patients all had sickle cell anemia, while two of the white patients had pernicious anemia, one had sprue and one had severe secondary anemia due to chronic pulmonary hemorrhage.

AVITAMINOSIS

Two white males, one negro male and two negro females died from beri-beri heart disease.

HYPERTHYROIDISM

Three negro males and one negro female died of congestive heart failure secondary to thyrotoxicosis.

PULMONARY DISEASE

Four negro patients died from heart disease secondary to pulmonary disease. Two females and one male had Ayerza's disease ane one male had chronic cor pulmonale secondary to pulmonary tuberculosis.

OTHER TYPES

This group included a negro female and a negro male with amyloidosis of the heart and a negro male developed acute dilatation of the heart secondary to a transfusion given too rapidly.

UNKNOWN

One case of diffuse, non-suppurative, isolated myocarditis and one case of acute cardiac dilatation were included in this group. Both were negro females.

TRAUMA

There was one death in a negro female due to a stab wound severing the anterior descending coronary artery.

COMBINED

This group contained five cases showing two or more etiologic types of heart disease which all contributed to final death. Two white women, 23 and 20 years of age, had congenital and rheumatic heart disease complicated by bacterial endocarditis. One white woman, age 78, had a syphilitic saccular aneurysm of the aortic arch which terminated in a dissecting aneurysm extending into the common iliac vessels. A white man, age 76, had rheumatic and syphilitic valvular lesions and severe coronary arteriosclerosis. A negro man, aged 55 years, had rheumatic, hypertensive, arteriosclerotic heart disease with fenestration of the cusps of the aortic and pulmonary valves and an aneurysm of the right coronary artery 2.5 inches in diameter.

SUMMARY

A statistical survey of 1,045 cardiac deaths occurring in the Charity Hospital of Louisiana at New Orleans between the years of 1935-1940, inclusive, has been presented and analyzed according to age, race, sex, and etiology.

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DISCUSSION

Dr. W. A. Sodeman (New Orleans): The study carried out is important to every one of us. We are continually talking about and estimating the kinds of heart disease we see in south Louisiana and have not had reliable figures. As usually estimated, rheumatic fever amounts to 10 per cent of the total for Galveston and other centers, but the figures for our own area have never been satisfactorily analyzed. This study therefore is an important one.

There are some things that Dr. Holoubek did not say that I know he did because I have read his report in one of the medical journals. The incidence of these various forms of heart disease depends upon the case load, and upon the number of white and colored beds in the institution. If these factors are not taken into consideration the figures do not mean anything. Such factors I know have been taken into consideration in the present study.

There has been a lot of discussion about rheumatic heart disease in the South. Last month in one of our staff meetings it was stated that there is an increase in frequency of rheumatic fever at the present time, that ten or twelve years ago the incidence of rheumatic heart disease was very low and that it has increased remarkably since that time, especially in the last year or two. One of the men from the North said you have to educate the boys from the South about rheumatic heart disease. This should not be true of boys from Louisiana. I have been teaching in Tulane since 1932 and I do not know of any time when we have not been able to show the chronic stages of rheumatic heart disease. We have the major kinds that you see practically all of the time. We are having more of it now. Forty per cent of our cardiacs are not rheumatic as in New England but we have always had a sufficient amount of material as far as rheumatic heart disease is concerned, to teach its recognition. I went to medical school in the northern part of this country. I saw more than I see here; more on the wards there than here, but there has not been any dearth of material in New Orleans as far as rheumatic heart disease is concerned. Dr. Holoubek's figures are taken from 1935 to 1940 when some have said we have not had much rheumatic disease; you see we have. For the last two years I am sure the incidence of rheumatic fever would be increased in comparison.

Another thing that impresses me in the figures for rheumatic disease is that the incidence in white and colored is comparable with an edge for the colored race. Most of the colored which we have seen are Louisiana products, discounting the idea that our rheumatic patients come from the East or North. Most of the colored people are from Louisiana and most of them have not been out of the state. They got their rheumatic fever here; it started here and they started here. The disease in its course and incidence is entirely a product of this state. I wanted to take this opportunity to impress on you the importance of Dr. Holoubek's figures in connecting some of the remarks made in past years about rheumatic heart disease and its incidence here.

Dr. A. A. Herold (Shreveport): The excellent paper to which we have just listened dealt with heart disease itself. It would be interesting in closing if Dr. Holoubek would give a comparison, not as to whether rheumatic heart disease is more common in other climates but whether other diseases such as scarlet fever, whooping cough and diphtheria are more common and therefore more heart disease attributable to that. If he would give us an idea as to comparative figures it would be interesting. It is not quite on the subject of his talk but would be of interest.

Dr. E. L. Wenk (Cloutierville): In Dr. Hunter's article of the current issue of the J.A.M.A., he stresses the absence of pain in myocardial infarction in the colored race. I have had several proved cases of myocardial infarction in colored persons in my private practice and pain has been marked in each case. I wish to ask Dr. Holoubek if he can give us any further information on this question.

Dr. J. P. Sanders (Shreveport): I would like to ask if there has been any change in the incidence or percentage of syphilitic heart disease in the colored race in the past few years. In Galveston, Texas, in 1926 the percentage of deaths in heart disease in the colored was higher than the percentage here. Obviously in the past years we have been putting on venereal disease campaigns and I wonder if this has had any effect on the result of heart disease.

Dr. Joe E. Holoubek (In closing): The autopsies performed at Charity Hospital are about three colored to about two white and that does not upset our statistics any; just about the same as incidence, two-thirds of the patients who died were colored and one-third white.

We did not go over the clinical records to ascertain pain or anything like that, so I can not answer the question of pain in the negro. Many persons with coronary occlusions came in dead. If the diagnosis could not be made at autopsy then we went to the clinical record and reviewed it but practically all of the diagnoses were made in the autopsy room, so I am unable to answer the question.

Rheumatic heart disease does occur down here but rheumatic fever in its clinical form probably is not seen as frequently. In reviewing some of the percentages found in other places, New England, Chicago and elsewhere, in Chicago the incidence is 26.2 per cent hypertensive; 24.1 per cent arteriosclerotic; 9.7 per cent syphilitic and 29.2 per cent rheumatic. Further north in the New England states there is 29.2 per cent hypertensive and 39.5 per cent rheumatic, 25.7 per cent arteriosclerotic and 3.9 per cent syphilitic.

The incidence of syphilitic heart disease is, of course, much higher here and in Galveston; farther North the incidence is lower because the colored population is not very high. The majority of statistics in the North do not separate the white from the colored.

I do not have recent statistics as to whether the deaths from syphilitic heart disease are decreasing. You must remember all of these were deaths due to heart disease. If a man died from syphilitic heart disease he probably had primary lesions years before. I can not give information as to whether or not this has decreased; it is still prevalent.

REPAIR OF CHOLEDOCHUS DEFECT BY HEPATICODUODENOSTOMY OVER VITALLIUM TUBE

MARSHALL L. MICHEL, JR., M. D.†
NEW ORLEANS

The incidence of operative injury to the common bile duct has increased during the last decade. This is a direct result of the increased number of cholecystectomies which are now being performed for gallbladder disease. In former years, when cholecystostomy was the most popular biliary operative procedure, there were less reported instances of injury to the choledochus.

However, it is now generally accepted that cholecystectomy is the operation of choice in acute and chronic cholecystitis. Therefore, common duct injury has become a greater problem.

Injury to the choledochus (actual severance, trauma resulting in stricture, or suture placed around the duct) practically always occurs in an attempt to perform simple cholecystectomy. There are three important reasons for this: (1) the failure of many surgeons to visualize clearly the common bile duct before clamping off the cystic duct and artery; (2) anatomic variations of the common, cystic and hepatic ducts and the hepatic and cystic vessels; (3) excessive traction on the fundus of the gallbladder by means of clamps (which produces angulation of the common duct).

Contrary to statements made by those who oppose frequent common duct exploration and drainage, injuries to the common duct occur more often during simple cholecystectomy than during choledochostomy. This indirectly proves the importance of visualizing adequately the choledochus when performing simple cholecystectomy.

There have been rare instances in which common duct injury has resulted during other operative procedures.[2] The most common of these is gastric resection for duodenal ulcer. It is easy to understand how the choledochus may become involved in adhesions from a long standing chronic duodenal ulcer and can possibly be injured in attempting to perform subtotal gastrectomy with actual removal of the ulcer. However, these cases are relatively uncommon as compared to instances of common duct injury during attempted cholecystectomy.

Recognition of choledochus injury at the time of the original operation is extremely important. If the surgeon is aware of the injury at this time, immediate repair is possible and is technically much less difficult than repair performed at secondary operation.

At the original operation, if the duct has been accidentally injured the severed ends of the duct are much easier to identify because of the absence of adhesions. Furthermore, the repair of the duct is less difficult because retraction and contraction of the cut ends of the duct have not yet taken place.

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†From the Surgical Service, Touro Infirmary, New Orleans, Louisiana.

Maes[5] has recently stressed the importance of immediate recognition of common duct injury with repair of the defect at the original operation. He has reported a case of complete severance of the common duct during a difficult cholecystectomy. The injury was recognized immediately and the duct repaired. A rubber catheter was used because vitallium tube of the desired size was not available. The patient made an uneventful recovery and has remained well.

Various sizes of common duct vitallium tubes should be kept available in all hospitals where gallbladder surgery is done. Vitallium tubes are considered to be superior to the rubber catheter in the repair of common duct injuries.[2] There is a greater tendency for precipitation of bile salts in the lumen of the rubber tubes than in the vitallium tubes. Rubber acts strongly as a foreign body in the tissues, whereas vitallium is relatively inert.

Furthermore, the rubber tubing usually passes into the intestinal tract relatively early. The vitallium tubes on the other hand remain in place for a longer time (in all probability some vitallium tubes are never passed) and therefore there is less chance of subsequent stricture. The rubber catheter, however, has one advantage over the vitallium tube. The former is flexible and can be more readily bent to fit almost any type of common duct defect.

The exact type of operative procedure which is to be used in the repair of the choledochus defects (either stricture or severance of the duct or actual accidental removal of part of the duct) depends entirely on the size of the defect and the availability of the distal (lower) end of the duct.

If the defect is small and both ends of the duct can readily be isolated and brought together without tension, simple end to end anastomosis may be performed over a vitallium tube.[2] If it is impossible to approximate the two ends of the duct for direct suture, the tube may be held in place by infolding the peritoneal structures of the gastro-hepatic omentum. This may be reinforced with a fascial graft from the rectus sheath (Lord and Arthur).[4]

When a large defect is present, and particularly when the distal remnant of the duct is very small or cannot be found at all, some type of anastomosis between the common hepatic duct and the gastrointestinal tract must be performed. Various procedures have been advocated. The hepatic duct has been anastomosed successfully to the stomach, to the duodenum and to the jejunum. Hepaticogastrostomy is often unsuccessful because of the marked motility of the stomach. Hepaticoduodenostomy is technically easy if the duodenum is adequately mobilized. The anastomosis is usually secure because of the proximity of the second part of the duodenum to the porta hepatica and because of the fixation and lack of motility of the duodenum as compared to the stomach.[3, 6, 7]

Direct anastomosis of the hepatic duct to the jejunum is technically an easy procedure, but the postoperative incidence of cholangitis renders such a procedure prohibitive. Cholangitis occurs after hepaticoduodenostomy and hepaticogastrostomy, but the incidence does not near approach that of direct hepaticojejunostomy.

In recent years, Allen,[1] Cole[2] and others have advocated an anastomosis of the hepatic duct to an isolated loop of jejunum, using a modification of the Roux procedure.

By inverting folds of the intestinal wall into the isolated loop, a pseudo-valve arrangement is brought about and reflux of the intestinal contents into the biliary tree is cut down to a minimum and the incidence of cholangitis markedly reduced (Cole).[2]

This procedure is certainly a sound one from an anatomical and physiological standpoint and offers a new solution to this difficult problem.

CASE REPORT

Mrs. J. G., aged 42, was admitted to Touro Infirmary on April 12, 1944, for an elective cholecystectomy. For several years she had suffered from the usual symptoms of indigestion, flatulence, upper right quadrant pain and intolerance of fatty foods. She had never been jaundiced and there was no history of biliary colic, fever or chills.

On April 14 a cholecystectomy was performed.

The surgeon was of the opinion that the cystic duct and the artery had been cleanly clamped and ligated. There were no peri-cholecystic adhesions and by clamped traction on the fundus of the gallbladder it was possible to deliver the gallbladder well up into the incision. The procedure was not particularly difficult. In closing the gallbladder bed it was recorded on the operative note that there was a slight, persistent bile drainage.

The patient was seen by me in consultation on the fourth postoperative day. At that time, she was markedly jaundiced, the abdomen was distended and there was generalized rigidity and tenderness, slightly more marked in the right upper quadrant. The temperature was 103°, pulse 130, and the general condition of the patient was poor. The clinical picture was that of a generalized bile peritonitis.

On April 18, an emergency celiotomy was performed. Exploration of the biliary region revealed that the common hepatic duct had been severed close to the junction of the right and left hepatic ducts. The distal end of the common duct could not be located. Due to the patient's poor condition, no attempt was made to perform any type of anastomosis. A whistle-tipped catheter was placed into the hepatic duct and the subhepatic region was drained with several Penrose drains.

The patient's postoperative course was relatively uneventful. She immediately developed an external biliary fissure and the jaundice subsided. Her general condition improved rapidly.

On June 17, 1944, a hepaticogastrostomy was performed. This was done over a small vitallium tube and was successful for a period of four weeks. However, after a bout of severe abdominal pain the patient again developed an external biliary fistula through the old operative incision.

On August 22, 1944, celiotomy was again performed. The severed hepatic duct was located without difficulty. The duodenal arch was reflected medially and a small remnant of the distal common duct was found in the retroduodenal space. This stump of the common duct had a ligature still around it and had apparently been ligated by mistake for the cystic duct. The defect between the two severed ends of the duct was bridged with a relatively long, thin, vitallium tube. The peritoneum of the free margin of the gastro-hepatic omentum was folded over the tube by means of interrupted sutures of silk in order to hold the tube in place. This procedure was successful for only a period of five days, after which time the external biliary fistula again developed.

On October 16, 1944, celiotomy was again performed. An anastomosis was made between the hepatic duct and second part of the duodenum after the latter had been mobilized. The anastomosis was performed over a short, funnel-shaped vitallium tube. The funnel end of the tube was placed on the duodenal side and the anastomosis was performed with interrupted sutures of silk. This anastomosis has been successful and the patient has been free of jaundice and external biliary drainage. However, she has suffered three mild attacks of cholangitis which has subsided spontaneously in a period of three to four days. She is in excellent health and has gained approximately 23 pounds in weight.*

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*This patient has recently returned because of severe recurrent attacks of cholangitis. There has been no jaundice or evidence of obstruction whatsoever, and it is believed that the passage of food material through the duodenum precipitates these attacks of cholangitis. In the near future, unless the attacks subside under conservative treatment, the patient will have a gastro-enterostomy performed with a surgical occlusion of the pylorus. Due to the fact that there is no evidence of obstruction of the hepatoduodenal anastomosis, it is not deemed necessary to perform the operation described by Allen, Cole and others.

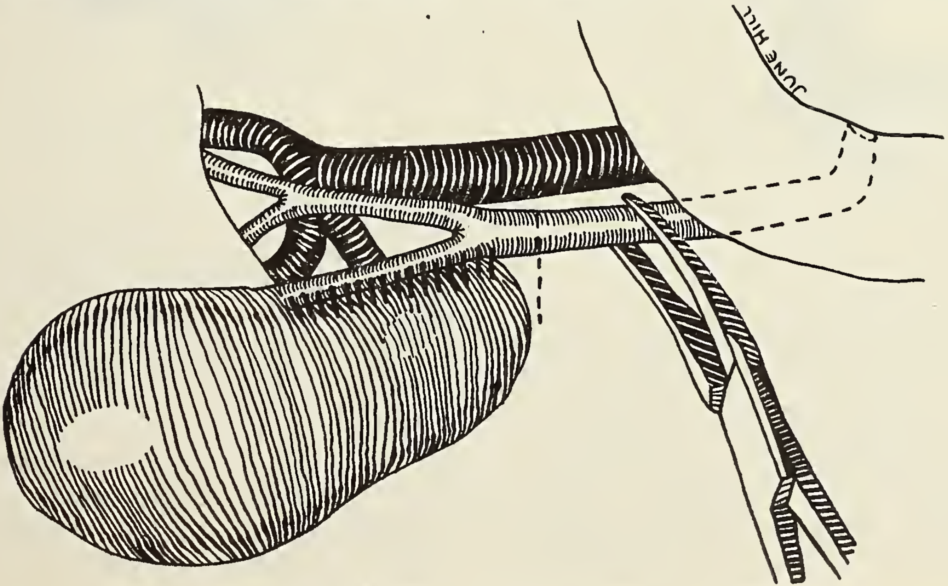

Fig. 1: Distended Hartmann's pouch adherent to cystic duct.

COMMENT

This case illustrates the complications resulting from accidental removal of a segment of the common bile duct in performing cholecystectomy. Furthermore, it shows the difficulties encountered when this error is not recognized at the primary operation. Persistent bile leakage into the subhepatic region during cholecystectomy is often indicative of hepatic or common duct injury and should always be carefully investigated from this standpoint.

There are a number of possible explanations of the choledochus injury in the case herein reported. In some instances, Hartmann's pouch is distended and adherent to the cystic and common ducts. The latter may be clamped by mistake for the former (fig. 1).

The cystic duct varies in length and occasionally runs parallel to the common-hepatic duct before joining the same to form the choledochus. If careful dissection is not carried out in such cases, either the choledochus or common-hepatic duct may be injured.

Excessive traction on the fundus of the gallbladder (fig. 2) may so angulate the choledochus that it may be clamped by mistake for the cystic duct.

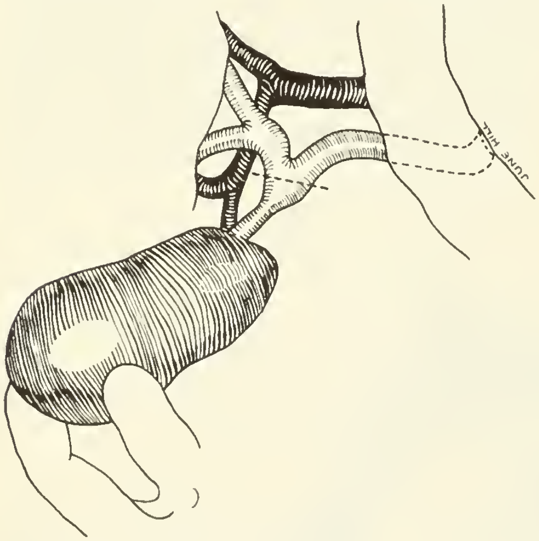

Fig. 2: Angulation of common duct by traction on fundus.

Bridging of the choledochus defect by means of a vitallium tube connecting the severed ends of the duct is the procedure of choice. This procedure more closely restores the normal anatomy. However, such a procedure is difficult if the defect is large, as in this particular case.

Hepatogastrostomy is a relatively simple technical procedure. However, the marked mobility of the stomach often will cause such an anastomosis to leak and eventually break down completely. This, I believe, happened in the particular case which is herein recorded.

An anastomosis of the hepatic duct to the duodenum was successful in this case, (fig. 3). Such a procedure is preferred by some surgeons (Sanders,[6] Dragstedt[3] because the bile normally enters the duodenum and the relative immobility of the latter renders stability to the anastomosis. However, cholongitis will sometimes occur and may cause postoperative difficulty.

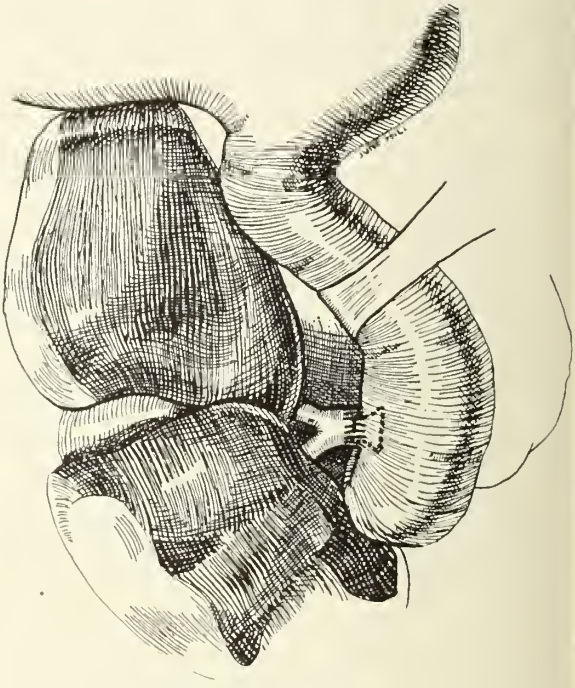

Fig. 3: Hepaticoduodenostomy over funnel-shaped vitallium tube.

Direct anastomosis between the hepatic duct and jejunum is not a difficult technical procedure. However, the high incidence of severe cholangitis renders such a procedure practically useless.

In recent years, Allen[2] and others have advocated the anastomosis of the hepatic and common bile duct to an isolated loop of jejunum. A modified type of Roux procedure is performed in order to isolate a loop of jejunum. The latter is then anastomosed to the biliary tree. Pseudo-valves in this loop of jejunum are then formed by infolding the wall of the bowel and the biliary tree is well protected against ascending infection from the gastrointestinal tract.[2] The incidence of cholangitis is practically nil after such a procedure.

It is entirely possible that this last operation may become necessary in the case herein reported, if the attacks of cholangitis become more severe and more frequent and if there is any evidence of inhibited hepatic function or obstruction of the hepaticodudenal anastomosis.

SUMMARY

1. An actual increase in the number of operative common duct injuries has occurred in recent years because of the increased number of cholecystectomies now being performed.

2. The high incidence of anatomic variations in the bile ducts and vessels predisposes some patients to common duct injury.

3. It is imperative to recognize a common duct injury at the primary operation in order to perform immediate repair. Greater technical difficulty is encountered at secondary operation because of extensive adhesions, presence of biliary fistula and retraction and contraction of the ends of severed choledochus.

4. Reconstruction of the common bile duct over a vitallium tube is the treatment of choice with end to end anastomosis.

5. Vitallium tubes are superior to rubber tubing because of the lesser degree of tissue reaction and deposits of bile salts therein. The vitallium remains in place longer and may prevent subsequent stricture formation.

6. When a large defect in the common bile duct is present, it is usually necessary to anastomose the common bile duct to some part of the gastrointestinal tract.

7. The various advantages and disadvantages of hepatogastrostomy, hepatoduodenostomy and hepatojejunostomy are discussed.

8. Recent reports indicate that an anastomosis of an isolated loop of jejunum to the choledochus is the most successful operative procedure in such cases.

9. A case is reported herein, in which a large part of the common bile duct was removed accidentally in performing cholecystectomy. Subsequent reconstruction of the common duct and a hepatogastrostomy was unsuccessful. Hepatoduodenostomy has been performed. The latter has been successful, but several mild episodes of cholangitis have occurred.

10. It is possible that re-anastomosis of the common duct to an isolated loop of jejunum may be necessary at a latter date in this particular case because of the complicating cholangitis.

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FEVER OF UNDETERMINED ORIGIN

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and

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NEW ORLEANS

Febrile illnesses are not uncommon in the South; even with the best of diagnostic facilities, many are undiagnosed. It is not at all unusual to see a patient with febrile symptoms (headache, malaise, vague somatic distress, nervousness, exhaustion, palpitations and weight loss), but no physical signs of a specific entity. Accordingly, the diagnosis "fever of undetermined origin" has become accepted generally. Needless to state, such a "diagnosis" is warranted only if the cause for the pyrexia is not evident after prolonged and thorough search for the cause.

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In the tropics, obscure fevers are frequent.[7] In the temperate zones these febrile states are encountered more than is generally realized. Sodeman[25] observes that what is the common cause of fever in one locality may be only an occasional etiologic factor in another, more remote site. Thus one would not necessarily search for the same causes of fever in New England as in the deep South. In similar fashion, in ascertaining the cause of a febrile episode in a man returned from overseas military duty, one would like to know what diseases prevail where the man served.

Within recent years rheumatic fever has become often suspected as the cause of an obscure febrile state. We know now that in a large proportion of cases of rheumatic fever, the classical signs are lacking. In Boston, as well as in New Orleans, studies have indicated that the majority of proved instances of rheumatic carditis are not preceded by arthritis, skin manifestations and chorea. Is it possible that many cases of "fever of undetermined origin" are due to an undiagnosed rheumatic infection?

The current interest in psychosomatic matters focuses attention on the question: Can fever be caused solely by a neurogenic or psychogenic process? Evidence to this end has been furnished by Reimann[24] and by Rappaport.[29] The latter observer reports a series of cases in which there was disproportion between the oral and the rectal temperatures. The oral temperatures rose above normal, whereas the rectal level constantly remained normal. This condition, which he calls "essential oral hyperthermia", was unaccompanied by any evidences of organic disease; in each instance, he attributes this thermal paradox to instability of the heat regulating mechanism in psycholabile individuals.

The purpose of our study was to determine the eventual outcome of a series of cases whose hospital diagnosis was "fever of undetermined origin." Similar studies (Alt and Barker,[2] Hamman and Wainright,[10] Keefer,[13] and Kintner and Rowntree[14]) have attested to the desirability of reviewing such cases. Since all our patients had their daily temperature variations recorded by oral methods, we have adhered to the standard that anything over 99.8° F. constitutes fever.

CAUSES OF FEVER

The heat controlling and heat regulating centers are believed to be located in the caudal part of the lateral hypothalamus and are apparently influenced in two ways—reflexly from the skin, and by the temperature of the blood flowing through them. The centers control body temperature by the transmission of sympathetic impulses to the cutaneous vessels, sweat glands, and pilomotor muscles. The thyroid and adrenal glands play their roles in the regulation of body temperature through the calorigenic effects of their secretions. Since the body temperature represents the balance between heat production and heat loss, a disturbance in this balance will be followed by a temperature change.

Carr[5] cites Leschke's classification of fever as due to:

(a) Physical changes of the surroundings—exhaustion or inadequacy of the heat dissipating mechanisms, as in heat stroke.

(b) Injuries to nervous centers—especially lesions of the following regions: third ventricle, internal capsule, medulla, or upper part of the spinal cord.

(c) Drugs and other chemical substances —dinitrophenol, large doses of adrenalin or thyroxin.

(d) Substances which arise from the destruction of body cells with liberation of toxins.

(e) Substances which arise from pathogenic microorganisms in an infected body.

If the cause of fever is not found by complete general and regional examination, it is well to consult the table of possible causes as suggested by Albrecht,[1] Harrell[11] and Keefer.[13]

I. SPECIFIC INFECTIONS
  - A. Septicemia (anaerobic streptococci, brucella, neisseria, eberthella typhosa, *Hemophilus influenzae*, pasteurella tularense, staphylococci).
  - B. Secondary syphilis
  - C. Malaria
  - D. Generalized infections
    1. Rickettsial group
    2. Rheumatic fever
    3. Relapsing fever
    4. Rat bite fever
    5. Trichinosis
    6. Lupus erythematosus
    7. Periarteritis nodosa
    8. Infectious mononucleosis
    9. Tuberculosis
  - E. Localized infections
    1. Renal and perirenal
    2. Hepatic (amebiasis)
    3. Subdiaphragmatic abscess
    4. Bronchiectasis
    5. Osteomyelitis
    6. Cholecystitis
    7. Thrombophlebitis
    8. Prostatitis
    9. Sinusitis
    10. Dental infections

II. TUMORS AND MALIGNANCIES
  - A. Generalized carcinomatosis, especially with liver metastases

III. DISEASES OF THE BLOOD FORMING ORGANS
  - A. Lymphomas
  - B. Leukemias
  - C. Pernicious anemia

IV. DISORDERS OF THE HEAT REGULATORY MECHANISM: METABOLIC DISORDERS
  - A. Hyperthyroidism

V. MISCELLANEOUS
  - A. Drug reactions
  - B. "Psychogenic" fever
  - C. Malingering

PROCEDURES SUGGESTED FOR STUDY OF OBSCURE PYREXIA

Blood—Complete blood picture, sternal marrow smears, studies for malaria, blood chemistry and Wassermann, repeated blood cultures (special media and anerobic conditions) agglutination studies, and sedimentation rates.

G. I. Tract—Gastrointestinal x-ray series, barium enema, proctosigmoidoscopy, stool studies for ova, parasites, and bacterial pathogens. Hepatic function tests, and cholecystograms.

Cardiorespiratory—Sputum studies, x-ray of the chest, electrocardiogram, bronchoscopy.

Genitourinary Tract—Urinalyses, prostatic secretion studies, cystoscopy and pyelography.

Other Tests—Lumbar puncture, x-ray studies of sinuses and teeth, basal metabolic rate determinations, biopsies (lymph gland, liver, skeletal muscle, skin, cervix).

CASE SERIES

Our cases were selected from 66,565 admissions to the Touro Infirmary between January 1, 1940, and December 31, 1944. All records with the primary diagnosis of "Fever of Undetermined Origin" were studied. These numbered 189, and from them there were chosen 45 records of patients 13 years of age or older with obscure pyrexia lasting at least 10 days in which no definite diagnosis had been made after thorough studies during hospitalization.

Follow-up letters were sent to the physicians who had treated these patients and subsequent data were obtained on 36 of the 45.

FOLLOW-UP OF 36 CASES OF FEVER OF UNDETERMINED ORIGIN

In only four cases was a definite diagnosis ever established. Of these three have died, and one was living and well when last seen three years after hospitalization for obscure pyrexia. These cases are reported in abstract below.

In 32 cases no definite diagnosis was ever established. Of these, 26 are living and well at the time of writing this paper. Four were living and well when last seen one and a half to two and a half years after admission for fever of undetermined origin. One is living and still ill (case abstract below). One is living and has carcinoma which is not considered the cause of his previous pyrexia (case abstract below).

ILLUSTRATIVE CASE REPORTS

In addition to the abstracts of six cases mentioned above we are reporting a case summary of a patient with fever seen here in 1945 in whom the ultimate diagnosis was Hodgkin's disease, and two cases from the senior author's practice. One illustrates recovery from recurrent bouts of fever without any diagnosis ever being reached. The second ultimately proved to be probably one of subacute bacterial endocarditis.

Specific infection: endocarditis diagnosed at autopsy.

A 49-year old white male was admitted January 29, 1941, with a history of fever of one month's duration. Past history revealed only "typhoid fever" at the age of 23. Examination showed pyorrhea and a loud, rasping mitral systolic murmur. During six weeks of hospitalization he had intermittent type of fever with almost daily elevations to 101 or 102° (F.). Five blood cultures, repeated malaria smears, sputum studies, agglutination (typhoid-typhus-melitensis groups) reactions, complete blood pictures, urinalyses, and serology, all were non-contributory. X-ray studies demonstrated normal sinuses; the chest showed increased pulmonary vascular markings, a slightly diffusely dilated aorta with calcium deposition in the arch, and the residuum of an old minimal (arrested) tuberculous lesion at the level of the right first interspace. Nothing suggesting active disease was seen in the chest plate. An electrocardiogram was abnormal (PR 0.12 seconds, QRS 0.06 seconds, sinus tachycardia rate 180 per minute, T-1-2-3 low or flat, S-1-2-3 slurred). Clinical impressions were questionably active rheumatic endocarditis or subacute bacterial endocarditis. Treatment with sulfanilamide and pyramidon gave no results other than temporary afebrile periods. On March 16, 1941 he was transferred to another hospital, where he expired in July, 1941. Autopsy showed large verrucous vegetations on the aortic and mitral valves; cultures from these vegetations were negative.

A 75-year old white male was admitted April 20, 1942 complaining of fever for 10 days, earache, sore throat, and cough. Cholecystotomy in 1939 represented the only previous operation. Examination showed no significant findings. During three weeks of hospitalization there was an irregular, intermittent fever exceeding 100° (F.) at times. The total white cell count was 14,000 (84 per cent neutrophiles, 3 per cent eosinophiles, 11 per cent small lymphocytes, 2 per cent monocytes); urinalysis, sputa, serology, malaria smear, blood culture, agglutinations (typhoid-typhus-melitensis-tularemia groups) all were negative. An electrocardiogram was within normal limits. Three x-ray studies of the chest at weekly intervals revealed arteriosclerosis of the aorta, osteoarthritis of the spine, increased right lung markings, and some enlargement of the right hilus; no gross infiltration was seen. The radiologist could not differentiate between interstitial pneumonitis or pulmonary congestion, and advised bronchoscopic study, which the patient refused. ENT consultant reported ears, nose, sinuses and throat as negative. Tentative diagnosis was carcinoma of the lung. No further studies were made, the patient was given a course of sulfathiazol, and discharged home, still having low grade fever on May 11, 1942. He died a few months later; autopsy revealed bronchiogenic carcinoma.

Diseases of the blood forming organs: Hodgkin's disease diagnosed by biopsy.

A 55-year old white female was seen in Medical Clinic in May, 1945, complaining of chills, fever, and profuse sweating of six months' duration. She was admitted to the ward June 6, 1945, where further studies revealed only a past history of umbilical herniorraphies in 1933 and 1934; complete regional physical examination was negative. During this first study an intermittent fever of 101 to 103° (F.) was noted on four consecutive days. The total white blood count was 15,800 (70 per cent neutrophiles, no eosinophiles, 30 per cent small lymphocytes); four malaria smears, agglutination (typhoid-typhus-melitensis-heterophile groups) reactions, blood NPN and sugar, cephalin flocculation, icterus index, stool culture, and blood culture, were all noncontributory. Sedimentation rate was markedly increased—110 mm. per hour. She was discharged afebrile June 15, 1945, the etiology of her fever still uncertain.

She was readmitted August 1, 1945 with the same complaints of fever and weakness. Physical examination again was negative. During this second period of 27 days' study a typical Pel-Ebstein fever curve was noted; a recurrent, remittent pyrexia with range between 100 and 103° (F.) for four days, followed by an afebrile period of five days, and then a five-day cycle with a temperature range between 100 and 103° (F.). Repeated search for evidence of lymphadenopathy, splenomegaly, or hepatomegaly was unsuccessful. The leukocytes numbered 12,800 per cubic millimeter (differential as on first admission). Sedimentation rate was 115 mm. per hour. Repeated malaria smears, blood cultures, stool cultures and examinations for ova and parasites, and a brucellergin skin test, all yielded negative results. The urologic consultant found a urethral stricture and a low grade cystitis, which were treated successfully and were not considered to be the cause of the fever. A KUB and a chest plate were negative. Emetine was administered for a possible hidden amebic hepatitis; penicillin was also given. No effect on the fever was noted. The impression was possible Hodgkin's disease. On August 28, 1945 she was discharged to the medical clinic. There a trial of atabrine and quinine was administered but without effect on the febrile recurrences.

In about December, 1945, in another city, the patient first became aware of a mass in her left axilla. She was admitted to another clinic, where the lesion was excised. The report of the pathologist was forwarded to us, and confirmed Hodgkin's disease.

The patient returned to the Touro Out-Patient

Clinic in late December, 1945, and has subsequently received a series of deep x-ray treatments for a group of enlarged glands in the left supraclavicular region. At present (March, 1946) she continues to have recurrent bouts of fever, has a negative chest plate, and no other signs of lymphadenopathy, splenomegaly, nor hepatomegaly. There has been no weight loss.

Disorders of the heat regulatory mechanism: hyperthyroidism.

A 29 year old white female was admitted May 20, 1941 complaining of low grade fever, chills, weakness, loss of appetite, and weight loss, of four to five months' duration. Past history was noncontributory and examination (including pelvic) revealed only a congenital erosion of the cervix. During the three weeks of study her temperature revealed a low grade, irregular fever of between 99 and 100° (F.), white blood count was 10,500 with 76 per cent neutrophiles and 24 per cent small lymphocytes. Urinalysis, serology, agglutinations (typhoid-typhus-melitensis-tularemia groups) stool and blood cultures, all added no information. A BMR was minus 6 per cent. X-rays of the chest and gastrointestsinal series were negative for pathology. She was given one blood transfusion (red blood cells 4,200,000 with 80 per cent hemoglobin on admission) and was discharged on June 13, 1941 still febrile, following courses of sulfathiazol and sulfapyridine.

One year later definite findings of thyrotoxicosis were found by her physician, a successful thyroidectomy was performed, and she was in good health when last seen in 1944.

Miscellaneous causes: cirrhosis of the liver. Diagnosis established on subsequent admission, with fatal termination.

A 57 year old white female was admitted on August 4, 1941 complaining of low grade fever, cough, and nausea for four months following a bout of "influenza." There was a past history of hematemesis in 1940 attributed to a possible duodenal ulcer. Examination showed only poor oral hygiene, hypertension, and a soft systolic apical murmur. During the four days of study there was a regular daily temperature rise to 100° (F.). The liver and spleen were palpable at no time, and no skin lesions were noted. Gastric analysis showed no free hydrochloric acid and nine degrees of total acidity. Blood NPN, sugar, CBC, melitensis agglutination and skin test, and serology were all negative. X-ray of the chest demonstrated a prominent left ventricle and a dilated thoracic aorta. A gastrointestinal series was negative. No treatment was given, no clinical impression was formed, and she was discharged still febrile on August 8, 1941.

She was readmitted on October 11, 1941 with a typical clinical picture of terminal liver failure and expired on October 27, 1941. At autopsy a Laennec's cirrhosis of the liver was found.

Recurrent fever more than three years: Definite diagnosis has never been established.

A 13 year old white female was admitted on July 29, 1942 complaining of fever of 10 days' duration. There was a past history of recurrent "pyelitis" of "12 years' duration." The patient attributed the present bout of fever to an injection of typhoid vaccine received the day the pyrexia began. Examination revealed an acutely ill, somewhat lethargic girl; the nasopharynx was injected, the spleen just palpable, and there were a few moist rales at the left lung base. During the five weeks of study a septic and remittent type of fever was noted almost daily, ranging from 100 to 103° (and at times to 104°) (F.). Repeated CBC's showed a WBC range usually under 10,000; the lowest was 4,800 and the highest 12,500. The differential count typically was: 72 per cent neutrophiles, 3 per cent eosinophiles, 22 per cent small lymphocytes, and 3 per cent monocytes. Urinalysis showed a few casts and sulfathiazole crystals on admissions, but repeated specimens (including catheter specimens) were negative. Blood agglutination (typhoid-typhus-melitensis-dysentery groups) studies were repeatedly negative, as were stool and blood cultures. Spinal fluid pressure on admission was 40 cm. water; a later one, 155 cm. water, but chemical, serologic, and bacteriological studies of these specimens gave no positive findings. X-ray studies of the chest and abdomen were negative. Electrocardiograms showed tracings within normal limits (P-R intervals 0.15 and 0.13, QRS intervals 0.07, right axis deviation-slight). An EENT consultant found no abnormalities. During the period of hospitalization, the lethargy cleared. A faint "butterfly rash" was noted by several observers over the bridge of the nose. There was no response to courses of quinine and of sulfadiazine. The febrile course persisted; on the day she left the hospital (August 26, 1942) a faint pericardial friction rub was first noted. Clinical impressions were: possible disseminated lupus erythematosus, miliary tuberculosis, or virus encephalitis.

Her physicians report (March, 1946) that she is still alive, has had a chronic febrile course over subsequent years, has been treated by several other physicians, and that no definite diagnosis has ever been established.

Presumptive diagnosis: subacute bacterial endocarditis—two cases.

Mrs. E. F., 41 year old white widow, reported for examination August 25, 1945 because of fever of 18 days' duration. This fever tended to rise as high a 103°, usually in the afternoon but at times in the morning, and was accompanied by profuse perspiration, malaise and exhaustion. As a rule, it would subside after three or four hours at the end of which time the patient would feel well enough to undertake her customary household duties. The single physical finding of note in the afebrile phase was a systolic apical murmur. Since one of her brothers had died of subacute bacterial endocarditis, the suspicion of this illness was constantly entertained. Repeated blood cultures were negative by the aerobic and the anaerobic methods. On at least six occasions, attempts were unsuccessfully made to demonstrate agglutinins against the various strains of brucella, typhosus, paratyphosus, tularense and B. Proteus OX 19. Samples of blood were submitted to various laboratories, including that of the National Institute of Health, for these determinations; none was ever recorded as positive. Blood was injected into guinea pigs; no pathologic process was demonstrated. Examinations for such rare conditions as rat bite fever were conducted with negative findings. Thick and thin smears for malaria plasmodia were negative. X-ray examinations of the chest, the teeth, accessory nasal sinuses, gastrointestinal tract were all within normal limits. No blood dyscrasia was demonstrable. Dental, urological and gynecological examinations were negative. Her fever continued in this fashion for about four months until one physician empirically administered 3,000,000 units of penicillin following which the fever and febrile symptoms disappeared and have not recurred to date. It is questionable whether this should be recorded as an instance of fever of still undetermined origin or whether one should accept it as a case of subacute bacterial endocarditis with no demonstrable bacteremia or skin manifestations, yielding to a relatively small amount of penicillin.

A. R. was a single, 28 year old white girl when first seen February 15, 1945. At this time she complained of painful and occasionally swollen ankles and intermittent fever. There was a history of rheumatic fever at the age of 12 years with incapacitation for one year. The details of this illness were unfortunately not available. She was pale, had a hypochromic anemia and there was a loud systolic and presystolic apical murmur. There was no x-ray evidence of pulmonary disease or of cardiac enlargement. Despite repeated attempts at blood culture, none was reported positive. The fever subsided after several weeks, the diagnosis "fever of undetermined origin" being made. Her symptoms did not respond to bed rest, large amounts of iron, and a high calorie, high vitamin regimen. She left the city for a period of two months' rest in the country and returned September, 1945, feeling much better and reporting "only occasional" fever. Again attempts were made to find the cause of the fever without success; she did not report again until September 15, 1945 at which time the fever had become steadily 103°. She was admitted to Touro Infirmary where a most extensive investigation was conducted without results. She awoke one morning to find some petechiae on the abdomen and on the hands and legs. Penicillin was begun in large amounts, and she received a total of 12,000,000 units in the suceeding two months. The fever receded somewhat and she was temporarily improved by repeated blood transfusions. At her own request she was discharged December 1, 1945. She was readmitted December 23, 1945 because she had awakened to find herself speechless and unable to move her right lower extremity. There were several new petechiae and the spleen was not palpable. The diagnosis of subacute bacterial endocarditis was made despite the continued negative blood culture. Sulfadiazine was given by vein and penicillin (total 18,000,000 units) by various routes. She soon made a speedy recovery and was allowed to go home February 28, 1946. She was afebrile for three months until following a dental extraction, her fever recurred and has persisted to date. There has been marked mental deterioration since this time and her general status closely approaches that of the senile person who has suffered a cerebral accident.

Undiagnosed fever: unrelated carcinoma developing eventually.

A 68 year old white male was admitted February 7, 1940 complaining of fever for about two and a half weeks, slight cough, some vague chest discomfort, anorexia, constipation, and mild abdominal fullness. There was a past history of typhoid fever at the age of seven years, tuberculosis at 28 years, and of known prostatic enlargement. Physical examination gave no remarkable findings, other than moderate prostatic enlargement. During the 10 days of hospitalization an irregular, intermittent type of fever was noted with rises of 100 to 101° (F.) almost daily. He had slight normocytic anemia, white blood cells of 25,000 and later 10,500 with 91 per cent and 74 per cent neutrophiles (respectively), and negative smear for malaria. Blood culture, serology, agglutinations (typhoid-melitensis groups), blood NPN and sugar were noncontributory. The genitourinary consultant found grade 3 benign prostatic hypertrophy, but after study of the urine and KUB could find nothing to account for the fever. X-ray of the chest showed no evidence of any active pulmonary disease; but there was hypertrophic osteoarthritis of the spine. Dental films revealed only alveolar resorption. After a short course of prontylin, the patient was discharged still febrile on February 17, 1940.

In April, 1943, he began to notice colicky abdominal pain and melena. A barium enema revealed a carcinoma of the middle third of the sigmoid in September, 1943. He was readmitted on December 5, 1943, and two days later an end-to-end anastomosis with resection was performed. The pathologist reported adenocarcinoma with metastases to the pericolic tissues. There had been no fever since soon after leaving the hospital in 1940.

At the present time he is living and comfortable. A barium enema in January, 1946, showed diverticulitis and irritability of the colon just proximal to the site of anastomosis, but no evidence of recurrence of the carcinoma.

DISCUSSION

The purpose of this study was to follow a series of patients whose febrile courses had not been satisfactorily diagnosed. It was appreciated here, as elsewhere in the domain of clinical medicine, that time is an essential factor in the diagnosis. If the patient is studied often enough and long enough, the diagnosis would become evident in most of them. In some instances, a diagnosis was established only at postmortem.

There still remained a group whose diagnosis was never satisfactory. What caused their "fever of undetermined origin"? The fact that 32 of the original group went without adequate diagnosis is impressive. Some of them had fever as long as two years without diagnosis of the cause. Several questions arise in our minds: How long can a patient continue to have fever without developing as an "interesting" phenomenon localizing diagnostic signs? How long can fever continue without curtailing a patient's physical abilities? How often do such patients spontaneously "burn out" their fever and cease to be patients? Now that powerful antibiotics are available, these questions are of more than academic interest. Are we justified in administering penicillin or streptomycin empirically to such patients, realizing that there is some risk in the development of a "penicillin-fast" state? Lastly the question arises: Is it possible that at least some of these instances are due to an unrecognized virus disease which eventually runs a self-limited course?

SUMMARY

1. Fever of undetermined origin has been briefly discussed with consideration of causes of fever, some of the more common disease entities ultimately diagnosed, and the procedure used in studying such cases.
2. Follow-up studies have been presented of 36 cases of obscure pyrexia.
3. Typical case abstracts have been given of undiagnosed, ultimately diagnosed and self-limited cases.

We wish to express our appreciation to members of the staff of The Touro Infirmary for allowing us to use their private case reports.

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CATARACT SURGERY AS DONE IN INDIA TODAY

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During the past winter I had the good fortune of being able to do eye surgery in India, and due to the transportation difficulties I was the only American over there doing eye surgery. This afforded me the unusual opportunity of working with the three leading cataract surgeons of India, Drs. Holland, McPhail and Mathra Das. These three men do more surgery than all the other eye men in India combined. I spent about six weeks with each of these men and performed over one thousand cataract extractions under their supervision. In addition, I observed over two thousand cataract extractions performed by these and other leading surgeons. I visited eye hospitals and talked to surgeons in Egypt, Arabia and Ceylon as well as in the larger towns and cities of India, and in all my travels I did not find two men using the same technic.

Few patients are seen before the day of operation, and then only a few hours before operation. No blood pressure is taken, no smear is made, and there is no chest or urine examination. The eye is inspected and if there is no apparent pus in the sac and the trachoma, which is present in approximately 97 per cent, is not too acute, and there is apparent glaucoma, (Sir Henry Holland takes tension after the patient is on the operating table), the eyes are operated on. If the eyes are glaucomatous an iridectomy is done, followed by removal of the cataract in three to ten days, depending on the operator. The majority of patients, however, are not operated upon if they can count fingers at twelve inches. Ninety-five per cent of these people are unable to read or write. Their chief requirement is to be able to distinguish a cow from a goat and to be able to walk about some. Of those operated upon, less than 10 per cent will get a plus ten sphere with no reading segment after operation. Therefore, if they can count fingers before operation, they are not satisfied with the operative results.

The patient is put on the table wearing the same dirty clothes in which he slept the night before. If a gown is used by the operator, it is for the protection of his clothes. No sterile piece or eye sheet of any kind is used. The eye is douched by an assistant with 1/2000 perchloride of mercury solution. The surgeon scrubs his hands with soap and water before starting the operations and not again unless he has to handle something other than the patient's eyes. He passes from one to the other but he uses sterile instruments for each eye.

Hospital beds, as we know them, are never seen except in a few hospitals in the largest cities where their total number of cataract extractions for twelve months is only a few hundred. A few fortunate patients get charpoys (rope cots) to sleep on but the average patient has some rice straw on the floor of a warehouse or on the ground under the trees. I have seen over 400 patients lined up in long rows on straw beds in a large warehouse. The patient is told to lie quietly for 24 hours but as the only nurse is a member of the family or a friend, he usually does more or less as he pleases in regard to nature's demands. The patient is allowed to go home about the tenth day. Surprising as it may seem under these conditions, infection (purulent endophthalmitis) occurs in only about one per cent of these cases.

As there is no standardized technic in either the capsulotomy or intracapsular method, I shall describe briefly the technic employed by each of the three surgeons with whom I worked, and also the different methods employed by other men with whom I visited.

Sir Henry Holland, who needs no introduction, has performed approximately 100,000 cataract operations in his hospital. About half of his operations are intracapsular; the other half capsulotomy. His criteria for the intracapsular method is an uncomplicated cataract in a calm patient over 50 years of age, not plethoric, without a bulging or "ox eye" and with the tension

(McLean) not over 30. If the tension is slightly raised, he does a posterior sclerotomy. He uses 4 per cent cocain and adrenalin for anesthesia, but if the patient is very nervous he performs the operation under general anesthesia, using chloroform.

In performing the intracapsular operation, Sir Henry Holland makes the incision entirely in the cornea from nine to three with no flap or bridge. He ruptures the capsule with the knife, often finishing the incision, and then does a broad iridectomy. An assistant keeps the eye fixed with forceps while the iridectomy is being done.*

In the capsulotomy method he uses the squint hood to express the lens while the spoon makes backward pressure on the scleral side of the incision, thereby causing a slight gap in the wound. On all patients he uses the O'Brien nerve block and also a suture in the upper lid which is taped to the cheek upon completion of the operation for the purpose of keeping the eye closed until the block has worked off. He operates on both eyes if indicated. The eye is dressed on the second day.

Dr. McPhail has performed some 50,000 cataract extractions in his hospital. He employs the capsulotomy operation exclusively, using cocain crystals for anesthesia. After the puncture is made, he ruptures the capsule with the point of the knife, then does the counterpuncture, and finishes the incision with a narrow conjunctival bridge about 4 mm. wide, slightly nasally, and then does a broad iridectomy by inserting forceps on the temporal side close to the bridge. He holds the speculum with the left hand and expresses the lens with a curved, glass lens expressor. The cornea is then massaged superior to the limbus with the lens expressor to remove any loose cortex. If the patient is very nervous he removes the speculum and delivers the lens by the same maneuvers, using the thumb on the brow and the middle finger on the lower lid to keep the eye open. Dr. McPhail does not have an assistant while performing this operation. The patient walks from the operation room and his eye is dressed on the third day.

Dr. Mathra Das, the Henry Ford of cataract surgery, has performed more cataract extractions than any man who has ever lived, and probably more than any other man ever will. Up to January 1, 1946 he had done 188,663 cataract extractions, and since that date he has operated upon an additional 2,000 or 3,000. His record for one day is 707 extractions. He uses four tables and his usual speed is one a minute if there are no interruptions. He holds "blind camps" all over India and Burma and has from 200 to 2,200 cataract operations in each camp. The last day I spent with him before I came home, we did 237 cataract extractions in 270 minutes. I did about one-third of the operations.

Dr. Mathra Das uses intracapsular method in all patients over 30 years of age unless patients have "ox eyes" or are very unruly. He uses 3 per cent butyn for anesthesia. If mild trachoma is present he paints the lids with 12 per cent silver nitrate and then operates with surprisingly good results. He makes the incision entirely in the cornea, finishing the incision about half way between the limbus and pupillary margin. This leaves a corneal scar but the average patient does not complain about it. He claims that lens delivery is easier with this type of incision.

In performing an iridectomy, Dr. Mathra Das uses curved iris scissors without the eye being fixed. He removes the speculum and an assistant takes care of the lids. The upper lid is held away from the eye by a squint hook while the operator's left thumb pulls back on the brow. At the same time the left index finger steadies the hook which can be moved by him in any direction for better exposure. The lens is expressed with the point of another squint hook, the pressure being made well below the limbus upward and backward so that the lens is tumbled with the lower edge presenting.

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*Formerly Sir Henry Holland advocated performing this operation without having the eye fixed. However, he has changed his technic and now prefers to keep the eye fixed during operation.

If the capsule ruptures in this position, it is easily removed with forceps. The point of the hook follows the lens up, tucking the cornea under the lens, and if the lens sticks in the incision, the hook is pushed behind and the lens is lifted out. The patient's eye is dressed on the seventh day.

COMPLICATIONS AT OPERATIONS

Other than expulsive hemorrhage, accidents at the time of operation are directly proportional to the surgeon's ability. Vitreous loss is the most frequent complication in either method, and is about the same in each, 3 per cent. The loss of a few drops of vitreous does not seem to influence the outcome. All of these doctors claim that it does no harm.

Prolapse of the iris is a serious complication which occurs more frequently in the intracapsular than in the capsulotomy method, 3 and 1.5 per cent respectively. Some surgeons excise the prolapse at first dressing while others wait until the eye is quiet before doing anything. However, the tendency now is to wait about six weeks until the eye is quiet. The majority of these men do an iridectomy but there are no figures available to substantiate their claim of less prolapse. In the eye hospital in Colombo, where approximately 2,000 cataract extractions are performed yearly, it is claimed that the incidence of prolapse is 1.8 per cent less without iridectomy. In these hospitals iridectomy is done only when there is prolapse at the time of operation.

Expulsive hemorrhage occurs in about 0.5 per cent of these patients while on the table and is found in another 0.5 per cent at the time of first dressing. Of course, vision is always lost in these eyes.

Dislocation of the lens occurs so seldom with experienced operators, it is almost neglibible. However, many patients come in with dislocated lens which has floated up behind the pupil. Of course, these lenses have to be removed with the spoon. The lens couchers still do a big business in India.

Secondary cataract is a frequent complication following capsulotomy, and its treatment cannot be taken lightly. It necessitates puncture of the coats of the eye and is frequently followed by infection. In 86 needlings there was one infection. In 600 consecutive capsulotomies secondary cataract was found in 24.6 per cent at first dressing. Of course, some of these patients will have clear eyes in a few weeks, but since there is no way to follow these patients, the residual cannot be determined. In 400 consecutive intracapsulars, secondary cataract was found at first dressing in only 2 per cent. The tendency now is to remove the capsule with iris forceps instead of the conventional cutting or tearing operation.

Eversion of the cornea occurs in about 0.1 per cent.

Iritis is a frequent complication following capsulotomy but relatively rare following intracapsular. The percentage is approximately 15 and 2 per cent, respectively.

VISUAL RESULTS

As follow-up is impossible on the majority of these patients, it is impracticable to get any figures on the vision of the capsulotomy patients as compared with the intracapsular after a period of one, two, or more years. Very few patients are ever seen after they leave the hospital, and even while they are in the hospital the records are very meager and incomplete. If a patient can count fingers, his vision is recorded as good. No accurate refraction is done on the few patients who get glasses. A plus ten sphere is put on and if they say they can see better, that is what is prescribed. Perhaps 10 per cent of the operated patients get glasses.

CONCLUSION

1. More physicians in India use capsulotomy than the intracapsular method, but more cataracts are removed by the intracapsular than the capsulotomy method. There is no generally accepted technic in either method—each doctor is a law unto himself. (I saw about a dozen varieties of each method and even observed one man

who performed a canthotomy on each of his patients.)

2. Visual results, without second operation in a large percentage of these cases, are definitely superior in the intracapsular method.

3. With an experienced operator, loss of vitreous is about the same in either method but recovery is quicker and smoother in the intracapsular than in the capsulotomy method.

4. Expulsive hemorrhage occurs in about the same percentage of cases in either method.

5. Prolapse of the iris occurs more frequently in the intracapsular than the capsulotomy extraction.

6. Some surgeons claim that iridectomy increases the incidence of prolapse just as vehemently as the advocates of iridectomy claim that it reduces the incidence of prolapse.

7. With the skillful operator the intracapsular method seems to be the one of choice in uncomplicated, senile cataracts, producing results equal or superior to the capsulotomy method (except in the incidence of iris prolapse.)

8. One type of senile cataract is as easily removed by the intracapsular method as the other, provided the operator knows what type he is dealing with and makes his incision accordingly. There is greater vitreous loss when the conjunctival bridge is used due to the resistance offered to the passage of the lens.

9. Asepsis does not seem to play a very great part in the incidence of infection which is about the same in the better hospitals as in the camps.

10. One thing stands out clearly: The more experience a man has the less he complicates the operation by special instruments, unnecessary injections, questionable procedures and multiple operations such as preliminary iridectomies (other than in glaucoma), superior rectus suture, canthotomy, and so on.

THE PATHOLOGIC PHYSIOLOGY OF DIABETES MELLITUS*

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The average clinician can and does treat a diabetic patient adequately but, for the most part, on an empirical basis, having previously learned the routine to follow, the dosage of insulin to be administered, and the time of administration. However, the basic knowledge regarding this disease and its pathogenesis is severely lacking in these well-meaning physicians. It is true that even a Senior medical student might treat a diabetic in the routine manner and do a good job of it but, to support adequately and understand the condition in a patient, one must have the disease and its physiology within easy memory distance.

During the last month of my Junior year, while in medicine, I first realized my very inadequate knowledge of this condition and took upon myself the task of clearing up this situation in my own mind, and at the same time, write my Senior thesis. I apologize for the method of presentation, presenting no original work but actually a summary of a group of much more extensive articles by men who are responsible for present-day concepts of this interesting condition, diabetes mellitus.

I. *Normal Sugar Metabolism.* — Before we can discuss abnormal carbohydrate metabolism as seen in diabetes mellitus, a review of the normal must be considered.

A. *Digestion and Absorption.*—Following the ingestion of a carbohydrate meal, many reactions, not too well understood, take place. Wiggers[1] has prepared an excellent chart which shows graphically these changes.

All of the carbohydrate ingested soon assumes the form of monosaccharides. Of necessity, these must all later be converted to d-glucose by the liver in order that utilization of the carbohydrate may take place. It has been shown that no glucose is absorbed from the stomach.[2] The small intestine happens to be the site of the majority of the absorptive processes because of its greater length, large surface area, augmented by the 5 million tiny, active villi found as part of the wall, and the large lymphatic and blood vessel arrangement. Absorption is regulated, according to Best and Taylor,[3] by physico-chemical laws, such as osmotic pressure relationships between blood sugar and that in the enteric lumen, hydrostatic pressure of the lumen produced by intestinal contractions, osmotic pressure of the plasma proteins and cataphoresis (the flow of a substance from the anode to the cathode across a membrane). These laws cannot adequately explain carbohydrate absorption, in the light of experimental work done by Cori.[4] He showed the rate of absorption for each sugar to be constant, regardless of the concentration of that sugar in the enteric lumen. The rate for each sugar was shown to be different and, relatively, galactose, glucose, fructose, mannose, xylose, arabinose. Therefore, some vital activity is given to the epithelial cells of the intestinal villi. Absorption rates of the selective type have been shown to be stopped at a temperature of 0° C., at which temperature the sugars were absorbed purely in accordance with their diffusion rates, depending on the size of the molecule. According to Verzar and McDougal,[5] the absorption of glucose depends mainly on a phosphorylation process rather than an oxidative one.

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*Awarded the prize offered by the Professor of Medicine at Tulane University School of Medicine for the best thesis on a subject related to internal medicine.

B. *Distribution.*—Following absorption, the carbohydrate may go one of several pathways, (a) conversion to glycogen in the liver, striated muscle, cardiac muscle, brain, kidney, or other tissue; (b) oxidation by peripheral tissues: (c) conversion to tissue fats and stored as such; (d) retention in the blood stream for variable lengths of time, or (e) excretion with the urine by the renal mechanisms. About 75 per cent of the glucose absorbed from the intestine is converted to glycogen in the liver.[6] Glycogen is found in all cells but most abundantly in the liver, where it varies with the nutritive state of the individual.[7] Shipley and Humel[8] found this liver glycogen to be in two parts, one as glycogen alone and the other as a component of glycoprotein with blood glucose. The latter remains at a fairly constant level, any fluctuation occurring with the glycogen protein. Gylcogen found in the muscles, liver and other tissues of the body is known to be 10 times the amount of glucose found in the body of the human.[7] Since the blood glucose is the connecting link between the many processes, it seems only proper to consider this factor next.

C. *Blood glucose.*—Glucose is found to be evenly distributed between the cells and plasma of the circulating blood, indicating a cell permeability to this substance.[7] Somogyi[9] analyzed blood filtrates by several methods, following fermentation, and found 23-31 milligrams of non-fermenting reducing substance per 100 c. c. of blood. He found these substances, thought to be chiefly glutathione, ascorbic acid, and thioneine, to be for the most part, contained in the red blood corpuscles. The remaining 60-100 mg. per cent of reducing substance is fermentable and therefore glucose. Since the "saccharoid" level remains fairly constant, regardless of changes elsewhere, the total reducing value of the tungstic acid filtrate is clinically considered to be equivalent to the blood sugar.[10]

(1) *Origin of the blood glucose.*—As mentioned above, there is an exogenous source of sugar which is that absorbed carbohydrate from the gastrointestinal tract. There is also an endogenous source of blood glucose. Soskin[11] confirmed Mann's[12] original work, establishing the liver necessary for maintenance of the blood sugar. Hyperglycemic agents were shown to have no effect on the falling blood sugar seen in a liverless dog. Examination of the muscles of these dogs revealed a normal muscle glycogen, which Soskin considered enough evidence to conclude that no blood sugar was formed from muscle in the absence of the liver and that the liver was the source of glucose in the absence of eating or

other exogenous source. Cori,[13] in 1931, showed that muscle glycogen could contribute indirectly to the blood glucose by producing an excess of lactic acid, which diffused into the blood stream, was carried to the liver, and there converted to liver glycogen to be changed to blood glucose if necessary. Young[14] showed, following hepatectomy in dogs, a lowering of the blood glucose but no elevation of the lactic acid level. By actual measurements, he concluded that less than 10 per cent of the sugar was obtained from lactic acid. Therefore, muscle glycogen is used only for local needs and contractions. The absorption of glucose by the muscles is an irreversible process and the glucose absorbed is retained until burned regardless of the body's need. The glucose → lactic acid → liver glycogen apparently occurs only during strenuous work or anaerobic exercise. This will be discussed later in more detail.

(a) *Liver glycogen.*—Drury[15] has aptly said "The liver is to the blood sugar what the heart is to the circulation." Glycogen, according to Peters,[16] plays an administrative role, forming glucose when it is not needed elsewhere. Cori[18] in 1940, dialyzed liver extracts, removing the inorganic and organic phosphates and the coenzymes. When glycogen, inorganic phosphate, and adenylic acid were added, there was a breakdown of the glycogen seen with the formation of glucose. If either the inorganic phosphate or the adenylic acid was removed from the system, no glycogen breakdown occurred. With further experimentation, it was found that glycogen + $H_3PO_4 \leftrightarrows$ glucose — 1 — PO_4, a reversible reaction, requiring the enzyme phosphorylase for its initiation. The reaction is an anaerobic one, inhibited by phlorhizin, and requiring adenylic acid as coenzyme. This glucose — 1 — PO_4 is changed to glucose — 6 — PO_4 by the action of phosphoglucomutase. When either glucose — 1 — PO_4 or glucose — 6 — PO_4 is added to liver extract, glucose + inorganic phosphate is formed. The enzyme involved in the latter reaction is phosphatase. Therefore, the combined action of phosphatase, phosphoglucomutase, and phosphorylase is required for the simple reversible reaction: glycogen (liver) $\leftrightarrows$ glucose (blood). Kaplan and Greenberg,[18] using radioactive phosphorus, noted the changes in the acid soluble phosphates in the liver coincident with metabolic changes in carbohydrates. This work supported Cori's fairly well. Cori,[17] however, had previously noted an uptake of inorganic phosphates by the liver in the formation of glucose from glycogen. This reaction (glycogen $\leftrightarrows$ glucose) goes to the right in the presence of insulin deficiency, stimulation of the liver by epinephrine, thyroid hormone and others, and in hypoglycemia resulting from starvation. With complete insulin lack, as shown by Soskin and Levine,[20] the liver glycogen reserves are depleted. When this occurs, the body begins to compensate with the production of glucose from body protein via liver glycogen.

(b) *Protein.*—If the carbohydrate stores of the body become depleted, protein takes over the job of supplying the carbohydrate needed. If only decreased carbohydrate is present, the body uses very small amounts of it and only when necessary.

In 1890, Mering and Minkowski, using a depancreatized dog, showed glycosuria and quantitative excretion of any administered sugar. They concluded that no carbohydrate was being utilized. They found 2.8 grams of dextrose excreted in the urine for every 1 gram of nitrogen and established the present D/N ratio. Soskin[21] criticizes their technic and choice of animals for determining this ratio. He points out that a wide variety of D/N ratios were seen among the different animals used and also in the same animal, depending on the number of days since pancreatectomy and condition of the animal. Other experimentation, headed by Lusk[22] repeated the experiments and found the D/N ratio to be 3.6 instead of Minkowski's 2.81. Lusk went further by saying if there were 3.6 grams of glucose in the urine to every 1 gram of nitrogen, then $\frac{3.6}{6.25} \times 100 = 58$ grams of glucose for every 100 grams of protein ingested. Soskin[21] concluded that sugar was derived partly from protein but considered it impossible to say to what extent it occurred since some fat might have been converted to sugar also. Shaffer[23] considered the glucose ingested as 100 per cent CHO + 58 per cent P + 10 per cent F. Ten per cent of the fat weight is in the form of glycerol and this portion is or can be converted to glucose.[24] Young[14] gave fat to depancreatized and fasting animals and noticed no elevation of the blood sugar or glycosuria or rise in liver glycogen. While the possibility exists that the animals used were already using their maximum amounts of fat in producing blood glucose. Drury says that while it may be possible, the experimental evidence has yet to be shown. Stadie[25] concurs with Drury in saying the diabetic picture can be explained without the necessity of an hypothesis similar to this: that fat → carbohydrate.

Having reviewed some of the experimental evidence, we can say that, in the normal individual, the blood glucose is supplied entirely by the liver and any exogenous means which may be used. However, when this liver is depleted of some or all of its glycogen, the body supplies of protein "pitch in", so to speak, in an attempt to keep the body well supplied with glucose. The possibility of any of the fat molecule, excepting the glycerol fraction being converted to glucose is not yet shown to be an actual process.

(2) *Utilization of the blood glucose.*—The glucose present in the blood stream may be utilized, that is, disappear from the blood stream and extrahepatic tissues, by one of the following four means:[7]

(a) *Formation of glycogen.*—This may take place in the liver or muscles of the body. It has been demonstrated that the glycogen content of the liver increases following a carbohydrate meal.[14] Kostelitz and Ritchie[26] found the glycogen content of the liver to increase with glucose administration and insulin injections. The phosphorus content of the liver was similarly increased when the glycogen and glucose levels were raised. Mann[13] found that only glucose could be utilized by the peripheral tissues, all other carbohydrate having to be changed to glucose or a polymer by the liver. Therefore, since glucose will be used by both liver and tissues, while the others are used only by the liver, the glucose should disappear faster, as it has been shown to do. The process of glucose ⇆ glycogen has already been mentioned but I will again show it to impress further these reactions upon the reader. Glucose + inorganic phosphates, catalyzed by phosphatase → glucose — 6 — PO_4 → glucose + PO_4, with the aid of phosphoglucomutase → glycogen, catalyzed by phosphorylase. This reaction occurs in both liver and muscle, where glycogen is mostly laid down. Dorfman[26a] states that while evidence does not rule out other pathways, they are unnecessary to explain the facts. Formation of muscle glycogen differs from that in the liver by being an irreversible process in the muscle, while in the liver, the glycogen ⇆ glucose relationship is easily reversible by a number of factors. The glycogen in the muscle is removed only by oxidation, aerobic or anaerobic.

(b) *Oxidation in the tissues.*—This process may occur either with or without oxygenation. In 1907, Fletcher and Hopkins[27] working with frog muscle under anaerobic conditions, noted an increase in lactic acid in the muscle. Embden[28] later said that the lactic acid played no part in contraction of muscle and that it was formed after contraction was over as well as during contraction. Meyerhof[29] argued this point with him, saying it was essential to the muscle contraction and was the only source of energy during work. Eggleton and Eggleton discovered creatine phosphoric acid, Meyerhof and Lohmann[30] later showing that the breakdown of this substance in active muscle gave off heat. Lundsgaard,[31] in the early thirties, used iodo-acetic acid, which inactivated the enzymes necessary for lactic acid formation. The muscles examined under that condition were able to do work but less than the normal muscle; so the lactic acid was shown not to be essential to contraction but necessary for an energy reserve. Along with these same conditions, it was noted that the intensity of the breakdown of creatine phosphoric acid was directly related to the amount of work done until exhaustion took place, at which time, another substance, adenylic phosphoric acid, began to break down. Further experiments were done, most of them by Lundsgaard and his co-workers, until some definite conclusions were reached. The breakdown of creatine phosphoric acid, releasing about 0.3 calorie per gram of muscle, was greatest in the earliest part of contraction, gradually decreasing through a series of contractions. On the other hand, lactic acid formation from glycogen, liberating about 1.0 calorie per gram of muscle, was very minute at first and increased in breakdown as the contractions were continued. This showed Embden to have been right. The energy produced by each substance tended to balance with a common amount of energy throughout the muscle contraction. Lundsgaard[31] relates the reaction in the following way: Adenylic phosphoric acid breaks down (cannot be demonstrated in humans) and in some way is related to the initial constriction of muscle. Following this, creatine phosphoric acid breaks down, the energy liberated producing a resynthesis of the adenylic phosphoric acid from its breakdown products. As the breakdown of creatine phorphoric acid decreases, lactic acid formation is taking place and producing enough energy to resynthesize creatine phosphoric acid from the creatine and phosphoric acid present. The lactic acid formation continues until muscular action ceases. One must remember that the above reactions, with lactic acid formation, take place in anaerobic conditions. As soon as oxygen is added, the situation changes. Fletcher and Hopkins[27] showed this when oxygen was added to the anaerobic frog muscle. The lactic acid which had accumulated disappeared. Lundsgaard[32] said, "No reason can be found for accepting Meyerhof's interpretation of the Pasteur-Meyerhof reaction in aerobically working muscles . . . . still speak of the Pasteur Meyerhof reaction in aerobically working muscle, for, by this, we merely understand that the formation of lactic acid recedes in the muscles under aerobic conditions." It has been generally assumed that aerobically working muscle metabolizes carbohydrate only, but we know that heavy exercise may be performed on a fatty diet alone and with a "fatty" respiratory quotient.[32] Possibly fat is being used directly or is converted to carbohydrate and used.

According to Meyerhof,[33] under aerobic conditions, no lactic acid is formed, the pyruvic acid being completely oxidized to $CO_2 + H_2O$. One more glance at the chart will reveal that pyruvic acid and not lactic acid is the intermediary substance of most importance. The lactic acid formed, just to clear up this point, is resynthesized to glycogen but not in the muscle where it is formed. It must be carried by the blood to the liver where it is changed to glycogen.[13]

(c) *Storage as fat.*—Peters[16] shows where carbohydrate can form any of the fatty acids with one exception, that being small quantities of unsaturated long-chain fatty acids. Once converted, it may be stored in anhydrous form and having potentially 9 calories per gram. When needed for energy, they need not be reconverted to glucose but may proceed to the liver, change to ketone bodies, and be oxidized in a metabolic cycle in the peripheral tissues, mainly the muscles.

(d) *Excretion by the kidneys.*—Cori[34] found glycosuria to be present in animals only after enough sugar had been absorbed to raise the concentration of the sugar in both the blood and tissues to a certain level. The renal threshold for glucose varies somewhat with the individual but the average quoted figure is 160 ± mg. of glucose per 100 c.c. of blood. Any level over the amount results in glycosuria. John[35] mentions that the threshold may vary any place from 65 to 450 mg. per cent. He brought out a rather interesting point, that it is difficult for the glucose to pass the renal threshold but, once the penetration has begun, it continues even with a level of blood sugar below the normal threshold.

(e) *Respiratory quotient.*—Since many investigators have used the R.Q. as evidence of carbohydrate and fat metabolism, we should consider it and evaluate it. The R.Q. is the ratio of $\frac{CO_2 \text{ expired}}{O_2 \text{ inspired}}$ and, according to theory, equals one if pure carbohydrate is being utilized. If one molecule of carbohydrate is oxidized ($C_6H_{12}O_6 + 6\ O_2 \rightarrow 6\ CO_2 + 6\ HOH$) one can see that the amount of oxygen breathed or utilized is equal to the amount of carbon dioxide excreted. In the completely depancreatized animals, the R. Q. is in the neighborhood of 0.7, which many take to indicate pure fat metabolism, since more O_2 is required for the oxidation of one molecule and, therefore, the R. Q. is lowered. Young[14] points out that sugar is being utilized to some degree, even in the depancreatized animals, and, that being the case, the R. Q. is only a resultant of two oxidative processes. And, if the R. Q. of the depancreatized animals is 0.7, with small amounts of glucose being oxidized, the actual R. Q. of fat must be less than 0.7. Young therefore suggests we reconsider our interpretation of the R. Q. Soskin[21] goes further than this, stating that the R. Q. is only an index for the whole body. The brain can utilize only glucose, so its R. Q. must be 1.0. Therefore, any other substance in the body, giving an R. Q. lower than this will not be shown to any accuracy by the overall R. Q. which is what is determined. Soskin mentions that oxidative processes alone do not make up the R. Q. but that many transformations and resyntheses and partial oxidations, all taking place in the body, have definite effects on the total R. Q. So, the true R. Q. is only an equilibrium among all the metabolic processes in the body. Werthessen[36] showed this by training rats to eat their 24 hour food requirement in one to five hours and found their R. Q. to vary from 0.27 to 1.70. He apparently caught the metabolic processes "with their pants down", so to speak, before the equilibrium of 0.7 — 1.0 usually found could be established.

(3) *Control of the blood sugar levels.*—Relative changes in the production and utilization of the blood glucose may occur and one can easily see the effect on the level of glucose in the blood. Definite factors produce these changes.

(a) *The blood sugar level.*—There is definite evidence to show that the level of sugar in the blood stream is one of the most important determining factors in the control of its own level. Soskin and Levine[20] have shown that carbohydrate utilization in the peripheral tissues, in the absence of insulin, increased proportionately to the elevation of glucose in the blood stream. In a normal individual, with normal amounts of insulin, an increase in the blood sugar level will decrease the output of glucose from the liver and vice versa. In addition, an increase in blood sugar causes the reaction glycogen $\leftrightarrows$ glucose to move to the left and vice versa. Upon these facts is the glucose tolerance test based. It was shown by Soskin, Allweiss and Cohn[37] that the decrease in the glucose blood level following the initial rise was not due to an increased output of insulin with increased utilization. They depancreatized animals, administered constant amounts of glucose with the necessary amount of insulin required to keep the blood level constant and then performed a glucose tolerance test. The test revealed a normal curve, indicating insulin stimulation was not a factor in this test. However, if the liver was removed instead of the pancreas and the experiment repeated, a diabetic curve was obtained. This showed conclusively that the liver's conversion of glucose → glycogen, the inhibition of the reaction glycogen → glucose, and increased utilization of sugar by the peripheral tissues were all on the basis of the increased blood sugar. Insulin in certain amounts was found necessary for the reactions to occur properly,[38] but no increase over this certain amount was needed, the stimulus being the high blood sugar level alone. Further work, quoted by Soskin,[21] supports the assumptions mentioned below:

(a) Small amounts of added glucose → inhibit glucose output by the liver.

(b) Moderate amounts → inhibit glucose output and increase glycogen formation in the liver and muscles.

(c) Large amounts → inhibit glucose output, increase glycogen formation, increase utilization of glucose by the tissues, and increase peripheral storage of glucose in the form of fat.

We can summarize the above data by saying a definite relationship exists between the blood sugar level and production on one hand and between the level and utilization on the other.

(b) *Insulin.*—In the absence of insulin, Soskin[21] states that no decrease in the rate of sugar utilization by the extrahepatic tissue occurs, if the blood sugar level is high enough. The liver, in an attempt to keep this blood level high, shows an increase in glycolysis with an increased glucose production and decreased glycogen content. With the decrease in liver glycogen, an increase in gluconeogenesis occurs with further changes. Administration of insulin will stop all of these processes and revert the metabolism back to normal. How does it work? Insulin administration will lower the blood sugar, increase the muscle glycogen and, depending on the blood glucose level, will raise or lower the liver glycogen. Cori and Cori[39] showed that the increase in glycogen in the muscle did not occur in the adrenalectomized animal and attributed the increase to a stimulation of adrenalin by the insulin, with adrenalin's effect causing the increase in muscle glycogen. Soskin and Levine[20] show that insulin is not needed for carbohydrate utilization, which can continue normally if the blood sugar is high enough, so insulin cannot produce an increased oxidation. But they say insulin is a catalyst, allowing the same amount of oxidation to proceed at a much lower blood glucose level. This sounds fairly reasonable. Lavietes[40] shows that, where the blood level was previously high, insulin will decrease glucose formation by the liver, thereby enhancing glycogen deposition in the liver. In the case of insulin administration to the normal person, a low blood sugar will result with decreased peripheral oxidation and glycolysis will be stimulated. Therefore, the process glycogen ⇆ glucose, according to Lavietes is secondary, not to the blood glucose level primarily but to oxidation of carbohydrate in the peripheral tissues.

Soskin[21] concludes that, since glucose utilization and glycogen deposition can occur in the depancreatized as well as the normal animal but at a slower rate for that blood level and at a normal rate if the blood level is elevated, insulin simply modifies the relationship between sugar concentration and sugar utilization rates. Lavietes[40] points out that insulin does not inhibit the liver glycogen → glucose reaction directly but by increasing the oxidation of carbohydrate at a lower level of blood glucose, the need for the high level, produced by glycogenolysis in the liver is lessened, allowing gluconeogenesis to surpass glycogenolysis with a resultant increase in liver glycogen. The increase in muscle glycogen seen following insulin administration is thought to follow the increase in liver glycogen, producing a lessened need for gluconeogenesis from protein and thereby stopping the breakdown of muscle glycogen. It must be kept in mind that a therapeutic paradox may occur if too much insulin is administered,[40a] decreasing the blood glucose level, which stimulates the liver to glucose production and thereby starting the processes forward again, with a decrease in liver glycogen, increased glyconeogenesis, and decreased muscle glycogen. Shipley and Humel[8] noted in liver slices, with insulin administration, a decrease in the liver glycogen and an increase in glucose output. They concluded that insulin accelerates the glycogen → glucose reaction. But, this being *in vitro*, it seems more likely that the reaction may proceed in either direction depending on the amounts of carbohydrate oxidized in the peripheral tissues, which in turn depends on the blood level and the presence of insulin. Stadie[41] thinks insulin aids in the phosphorylation of glucose which makes it easier for the tissues to store or utilize it. Soskin[20] definitely states that insulin does not promote the oxidative breakdown of glucose but is primarily concerned with the regulation of the blood sugar level, affecting the output of sugar from the liver and its rate of uptake but not oxidation by the tissues. Summing up this evidence, one can say that, according to the present views, which are still incomplete, insulin is a cataclyst, which is necessary for the rapid phosphorylation of glucose to glucose — 1 — PO_4, the presence of which easily facilitates tissue oxidation and glycogen production in muscle and liver. These processes of tissue utilization and glycogen production may continue in the absence of insulin but require much higher blood sugar levels than when insulin is present. And, lastly, all of the other effects of insulin can be explained adequately on the basis of this increased phosphorylation process alone.

Haist[42] has shown that the insulin content of the pancreas is decreased in two different and opposite conditions: (a) fasting, fat feeding, and insulin administration will, by relieving the need for insulin, make the cells less active. Insulin administration in this condition will further reduce the content. (b) Partial pancreatectomy or injection of anterior pituitary extracts into dogs will, by increasing the blood sugar level and keeping the level high, stimulate the beta cells of the pancreas to exhaustion with later atrophy and degeneration. Insulin administration in this type would prevent the pathologic changes occurring.

Stimulation of the beta cells is said to result[42] from high blood sugar levels, low blood insulin levels, a minor effect produced by the central nervous system, and from stimuli originating in the anterior pituitary gland.

Factors other than those mentioned above are of importance in regulating the normal blood sugar level. Long[43] brings out the fact that there are hormonal influences in the body which influence processes concerned with glucose utilization and those which affect the formation of glucose and other carbohydrates from non-carbohydrate sources. These factors will be considered below.

(c) *Influence of the anterior pituitary gland.*—Many experiments, in regard to the effect of the anterior pituitary upon sugar metabolism, have been carried out. In a depancreatized animal with typical diabetes mellitus, the removal of the anterior lobe of the hypophysis will produce an almost complete remission of the signs and symptoms previously noted.[43-46] The hyperglycemia, ketosis, acidosis, and glycosuria disappear, while the nitrogen excretion by the kidneys is reduced, the elevated D/N ratio is lowered, and the survival time of the animal is lengthened. The anterior pituitary, apparently, in the absence of the pancreas, promotes either an increased production of sugar or a decreased utilization or both.[46] Therefore, the removal of the gland was very similar in many ways to the administration of a dose of insulin. The above shows that, with the removal of the organ there occurs a decrease in tissue breakdown to glucose or an increased utilization of the carbohydrate.

If the anterior pituitary is removed in a normal animal[43] the sensitivity to insulin administration is greatly increased, there occurs an increased resistance to the hyperglycemic action of epinephrine, glucose absorption is retarded, and glucose tolerance is retarded or normal, the latter depending on the animal used. In addition to these effects noted by Long[43], Houssay[44-46] mentions a decreased nitrogen excretion by the kidneys, hypoglycemia, a marked decrease in liver glycogen, and no change in muscle glycogen in dogs and rabbits. Here again the effects seen may be explained either on the basis of decreased breakdown of tissue or increased carbohydrate utilization.

If the anterior lobe extract is injected into normal animals,[46] a hyperglycemia and glycosuria occur if the animal has been previously well fed; nitrogen excretion in the urine increased, the R. Q. decreases, and the animal's sensitivity to insulin is greatly reduced. A postmortem examination of the animal will reveal a certain amount of degeneration in the islets of Langerhans of the pancreas. To explain these actions of the anterior pituitary extracts, we must consider other information from further experiments: (a) If the liver is removed before the anterior pituitary extract is injected, the hyperglycemia is not seen.[43]

A previous section of the splanchnic nerves will produce the same effects as hepatectomy. The liver, therefore, is stimulated in some way, possibly through the sympathetics to produce more glucose from glycogen and is directly responsible for the hyperglycemia seen following an injection of anterior pituitary extract. (b) The R. Q. is decreased following an injection of anterior pituitary extract. Long takes this to mean that carbohydrate utilization by the tissues is decreased. (c) The increased nitrogen excretion seen above is thought to be indicative of an increase in protein breakdown by the body. So, apparently, the anterior pituitary extract stimulates glucose production in the liver, decreases carbohydrate utilization by the tissues, and increases protein breakdown in the body. Houssay[46] gives the anterior hypophysis four actions in regard to carbohydrate metabolism: (a) hypoglycemic, (b) hyperglycemic, (c) glycogenetic, and (d) glycogenolytic.

It is possible and probable that this gland acts directly on carbohydrate metabolic processes or is it more probable that this action is extended to other organs in the body? The part the liver plays has already been mentioned. Long,[43] using dogs, showed the glycosuric and ketonuric effects of the injection of anterior pituitary extract. These were not prevented by the previous removal of either the thyroid or adrenal medulla but were definitely inhibited when the adrenal cortex was removed. If, therefore, this action is carried out by the stimulation of the adrenal cortex, injection of adrenocortical extracts should produce similar effects in the metabolism of glucose.

(d) *Adrenal cortical influence.*—If adrenocortical hormone (Kendall's Compound, E, C-11-17 oxygenated adrenal cortical steroid) is injected into a normal animal, the blood sugar increases, glycosuria results, and large amounts of nitrogen are extracted by the kidneys.[43, 47] If an experimental animal is made diabetic by pancreatectomy and then, following this, a bilateral adrenalectomy is performed, the effect is almost the same as if the anterior pituitary gland had been removed, that is, a remission of symptoms and general metabolic disturbances occur.[43] This will not happen if only the medullary portions of the glands are removed. These effects are thought to be due to an increase in protein catabolism with gluconeogenesis since (a) urinary nitrogen rises before urinary sugar excretion does and (b) the blood sugar level is much higher than would be expected if only due to an inhibition of carbohydrate oxidation or increased production of glucose from the liver.

The weight loss and urinary excretion products have been compared between a case of pancreatic and one of adrenal diabetes.[47] The changes were found to be about the same but, whereas insulin injections cured the pancreatic case, it had no effect on the adrenal case except in extremely high doses. Therefore, this "sugar hormone" of the adrenal cortex is theoretically directly antagonistic to insulin, thereby decreasing carbohydrate utilization and stimulating to the breakdown of protein, with an over-production of nitrogen and glucose resulting. The protein mainly affected is thought to be that contained in the lymphoid tissues of the body.

The pituitary gland, by acting through the adrenal cortex, produces the same effects plus the effects resulting from its stimulating action on the liver, increasing glucose production. The degeneration of the pancreatic islets seen following an injection of the anterior lobe extracts is thought to be due to an exhaustion atrophy, secondary to the sustained hyperglycemia present.[48]This pathologic process is preventable by a previous or simultaneous injection of insulin with the pituitary extract, thus indicating no direct toxic effects on the beta cells by the hypophyseal hormone.

(e) *Others Factors Involved*

(1) *Thyroid hormone.*—The effect of the thyroid as seen in cases of hyperthyroidism is to produce a hyperglycemia and glycosuria. Joslin and Lahey[49] found a diabetic glucose tolerance curve and glycosuria in about one-third of a series of hyperthyroid patients in 1943. This effect was found to

be absent in experimental animals following hepatectomy or a splanchnic section indicating an action on the liver, increasing glucose production. Althausen et al.[50] noted an abnormal galactose tolerance curve in hyperthyroid patients following oral administration of the sugar but a normal response to an intravenous injection. This led him to say the action of the thyroid hormone was on the phosphorylation mechanism in the intestional mucosa, increasing its action and thereby glucose absorption in cases of hyperthyroidism. No direct effect on the metabolism, other than a generalized increase in rate, could be found.

(2) *Adrenal medullary hormone.*—Injections of this substance produce a hyperglycemia secondary to an increased mobilization from the liver.[3] Muscle glycogen has been shown to decrease, raising the blood lactic acid level.[14] The lactic acid is carried to the liver, where it is converted to glycogen, later being changed to glucose (Cori cycle). Small amounts of adrenalin will decrease liver glycogen, slightly larger doses will decrease muscle glycogen and increase liver glycogen, while large doses reduce both the liver and muscle glycogen contents. This hormone is not thought to act except in exercise and emotional disturbances.

(3) *Posterior pituitary gland.*—This organ was thought by Burns in 1923 to have an antagonistic action to insulin since no change in the blood sugar level was noted when posterior pituitary extract was injected subcutaneously with insulin. Griffith[51] repeated these experiments and used the intravenous route in addition, noting a normal insulin response. He concluded the effect of the posterior pituitary extract to be local, causing marked vasoconstriction and thereby preventing absorption of the insulin.

(4) *Diet and nutrition.*—Blatner and Murphy[52] noted a lowering of the blood sugar following the injection of 180 grams of whole fresh liver equivalent to the effect seen with 10 units of insulin. Biskind and Schreier[53] noted the same effects but related them to the vitamin B complex contained in the liver used. They mentioned other works, showing fairly well that vitamin B1, along with riboflavin niacin amide, ascorbic acid, vitamin A and vitamin D, are all essential to carbohydrate metabolism. When the liver is damaged by a toxic drug, a diabetic glucose tolerance curve can be obtained.[54] Since the conversion of blood glucose → liver glycogen and the reverse depends on enzyme systems in the liver, any dysfunction of these enzymes will bring about changes in carbohydrate metabolism, expressed as hyperglycemia, glycosuria, and a delayed glucose tolerance curve.[55] This type of dysfunction is seen in cases of obesity with fatty livers and in patients with vitamin B deficiency.

(f) *Experimental work with carbohydrate metabolism.*—In addition to the large amount of experimental work mentioned previously, there has been some good work done recently which should be at least mentioned in a paper on carbohydrate metabolism and diabetes.

(1) *Phlorhizin.*—Following the administration of this substance, a glycosuria, hypoglycemia and decrease in liver glycogen all occur, in addition to other effects. This specific action is thought to be on the phosphorylating mechanism so essential to carbohydrate metabolism. By inhibiting this mechanism, phlorhizin prevents intestinal absorption of glucose, slows down or stops the glycogen glucose processes throughout the body and inhibits glucose resorption by the renal tubules. Lukens, Dolens, and Wolcott, quoted by Lukens[56] compared the effects of insulin with those of phlorhizin as is shown by the table on page 461.

(2) *Alloxan.—This* substance, which is an oxidation product of uric acid and, as yet, not known to be present in the human intermediary metabolism of uric acid, has been found to be definitely diabetogenic in character. A considerable amount of experimental work has been done with this substance in an attempt to learn something further about diabetes mellitus, and related problems.[48, 57, 58, 59, 60, 61] Goldner[48] presents an excellent summary of the work on this substance up to January, 1945.

Phlorhizin	*Insulin*
A. *Similar Effects*	
1. Reduction of hyperglycemia	
2. Restoration of hydropic islands of Langerhans	
B. *Different or Opposite Effects*	
1. Impairs resorption of glucose by kidney	1. No such action
2. Increases protein catabolism (N excretion)	2. Decreases protein catabolism (N excretion)
3. Increases ketogenesis	3. Decreases ketogenesis
4. Amount of food assimilated by diabetics is increased, decreased, or unchanged.	4. Food assimilation in diabetics is increased
5. Slight gain or loss of weight occurs	5. Marked gain in weight occurs
C. *Uncertain Effects, Common Assumptions listed*	
The following are probably secondary to a loss of glucose:	The following may include the primary action of insulin:
1. Decreased carbohydrate oxidation	1. Increased carbohydrate oxidation
2. Liver and muscle glycogen is usually decreased but may be increased by adding glucose or insulin	2. Increased liver and muscle glycogen in the diabetic.

Following an injection of alloxan in diabetogenic doses, a typical diabetes mellitus becomes evident in twenty-four to forty-eight hours, including polyphagia, polydipsia, polyuria, hyperglycemia, glycosuria, and weight loss. During the first twenty-four hours, however, there occurs a marked hypoglycemia with death, if glucose is not administered. If insulin administration is continued, animals may live and remain well for many months. Without insulin, they lose weight, become weak, very susceptible to infection, show hyperglycemia, hyperlipemia, fatty degeneration of the liver and death in six to seven weeks. Depending upon the animal used, ketosis, acidosis, cataracts, and retinitis may be demonstrated. Postmortem changes noted are beta cell necrosis in the islets of Langerhans with no evidence of local inflammation and, with large doses, fatty infiltration of the liver and glycogen deposition in the renal tubules. Degenerative changes in the basophilic cells of the anterior lobe of the hypophysis with some necrosis of the cells in the adrenal cortex have been noted by Thomas and Emerson.[57] The latter pathological changes were not prevented by previous adrenalectomy nor could similar changes be produced by injecting large amounts of adrenocortical extract into the animals. So, these changes were not secondary to hyperglycemia but appeared to be a direct result of the alloxan.

The insulin content of the pancreas per gram of tissue is decreased following alloxan administration.[48] The hyperglycemias seen following injections of anterior pituitary extract occurred before islet cell degeneration took place and the degeneration was therefore thought to be secondary to the increased blood sugar level, being prevented by concurrent insulin administration.[48] This is not the case with alloxan diabetes, in which the degenerative changes are not inhibited or prevented by concurrent insulin administration. Therefore, a direct toxic action of alloxan on the pancreatic islets must exist. If the animal has been previously adrenalectomized, the symptoms of alloxan diabetes disappear or fail to appear.[58] Apparently gluconeogenesis must be stimulated by the administration of alloxan.

The hypoglycemia seen in the early part of the syndrome is not seen if the animal has been previously depancreatized.[48, 59] Therefore, either the islets are stimulated to exhaustion and later atrophy or the degenerative processes release a large number of insulin previously formed within the cell walls. Since the beta cell degeneration occurs before the hypoglycemia, the second theory is supported by Goldner. Bannerjie[59] reviewed the literature on the subject, and with his experimental results, concurred with Goldner in thinking the hypoglycemia to be due to the process of degeneration with the release of preformed insulin followed by hypoglycemia. Once this phase has passed, however, the animal remains in a constant state of hyperglycemia as a result of the insulin lack now present.

(3) Thorogood and Zimmermann[60] bring out the idea of a second pancreatic hormone which opposes insulin, raising the blood level of sugar, promoting glycosuria, and preventing ketosis and coma in insulin deficient animals. Some interesting work has been done on this possibility, which we will not have time to discuss.

II. *Metabolic Disturbances as Seen in Diabetes Mellitus*

Diabetes mellitus is defined as a disturbance in carbohydrate metabolism secondary to a deficiency in the insulin supply of the body.[43] This may be an actual or relative deficiency but this paper is concerned only with an actual one. First, a word about the pathology associated with this disease. Long[43] noted a disproportion between the severity of the disease process and the degree of destruction seen in the pancreas at postmortem examination. Gomori[62] found no changes in some seven cases of long duration and marked changes in occasioned non-diabetics. To produce glycosuria in a dog, a least nine-tenths of his pancreas must be removed. Warren,[63] in 1937, mentioned that regardless of the severity of the disease, no prediction could be made with any degree of certainty as to what would be found at autopsy. He said that at least one-fourth of the pancreases showed no appreciable pathology. Engelhardt and Bruno,[64] in a series of 101 cases examined at autopsy, found only 17 pancreases with any pathologic findings. Warren and Root[65] summarized the pathological findings thusly, "Whatever the cause may be, it seemingly acts over a long period of time, perhaps throughout the duration of the disease. The pathology which we find in the pancreas at autopsy rarely represents the initial damage to the organ, but rather the result of a long struggle between the regenerative activity of the pancreas and the degenerative changes caused by the diabetogenic factor. The pancreas is not a static organ like the brain or myocardium, unable to repair itself after injury. . . . believe that this unfavorable course of the disease is not due to failure of the pancreas to regenerate but to continued injurious action on the organ by the causal agent, eventually overcoming the regenerative efforts."

If one understands in detail the normal metabolic processes associated with carbohydrate metabolism and the effects of administering insulin in animals and humans, the changes seen with insulin deficiency should be indeed obvious.

(A) *Overproduction vs. Underutilization.*—Since the level of the glucose in the blood stream depends on two factors mainly, that is, production and utilization, a hyperglycemia could result from an increased activity of the first facor or a decreased acivity of the second. The two theories regarding diabetes mellitus are classified in the same way, one saying the disease is a result of the inability of the body to utilize glucose in the absence of insulin and the other claiming that glucose is being utilized in the normal manner but that too much carbohydrate is being produced in the absence of insulin.

Minkowski first began the underutilization argument. Lusk and his co-workers said the completely depancreatized dog could not utilize glucose. Their beliefs were based on the following experimental results: (a) The non-protein R. Q. of diabetics was at a level precluding carbohydrate oxidation.. (b) Any administered sugar was quantitatively excreted by the kidneys. (c) An increase in the arteriovenous difference of blood sugar levels in muscles in a normal animal after glucose administration was diminished or absent in a diabetic. With these facts proved, the overproduction school had to say that carbohydrate was produced from fat in the body and that being the case, the R. Q. would only be an average between the R. Q. of fat → sugar in the liver (less than .7) and the R. Q. of sugar being metabolized in the peripheral tissues (more than .7). Soskin's[21] remarks about the R. Q. make it rather poor evidence for or against any theory. And, since the body was working with the maximum amount of glucose it would handle anyway, no wonder that the A-V difference did not increase or quantitative excretion of ingested carbohydrate take place.

Mann and Magath[66] in 1913 demonstrated that the removal of the liver from the depancreatized dog resulted in the development of hypoglycemia at the same rate and to the same degree as in the normal dog. Mirsky[67] found this to be true in his experimental work. Yater, Markowitz, and Cahoon[68] found carbohydrate oxidation to be occurring in a diabetic but at a subnormal rate, however. Soskin and Levine[20] showed that a diabetic animal with a hyperglycemia could metabolize the normal amount of glucose a non-diabetic could at a normal blood sugar level. Therefore the overproduction theory seems more tenable at the present time.

The source of the extra glucose has been shown to be the liver and through it, various pathways of gluconeogenesis. It is possible that glucose may be formed from protein and fat deposits in the body. Most authorities in general agree that protein may be converted to glucose but it is still a controversial point; the results indicate the formation of glucose from fatty acids and ketone bodies. Stadie[25] believes in the overproduction of sugar as a result of the body's inability to utilize sugar to its fullest capacity but definitely is against any such mechanism producing glucose from fat. He claims that the extra energy needed is obtained from the utilization of fatty acids by the body directly, without conversion to glucose being necessary. Yater, Markowitz, and Cahoon[68] combined both theories, which seems reasonable, and said the diabetic animal cannot utilize glucose as well as the normal one and in some way stimulates the liver to overproduce glucose from glycogen. When the liver glycogen stores become depleted, gluconeogenesis from lactic acid and protein takes place. The conversion of fat to glucose is still unproved.

B. *Hyperglycemia.*—Accepting the last few statements and again referring to Soskin and Levine,[20] one can see what might be expected to occur in the presence of a lack of insulin. The tissues of the body cannot utilize glucose at the normal blood sugar level and, in some way, stimulate the liver to produce larger amounts of glucose, even with a normal or elevated blood glucose present. This elevation in blood glucose, if high enough, enables the tissues to utilize the carbohydrate normally. With the decreased amount of insulin present, the inhibiting action of insulin on glucose production by the liver is also reduced, and glucose is produced freely. The liver continues to supply glucose to the blood stream with little or no replacement of its depleted glycogen. When a certain point is reached, however, gluconeogenetic processes are speeded up in an attempt to replace the liver glycogen from protein and lactic acid stores in the body. As a result of the increased protein breakdown to form glycogen in the liver and the muscle glycogen → lactic acid → glycogen in liver, there occurs a marked weight loss. Mirski[67] has shown that insulin decreases the rate of glyconeogenesis from protein. Polyphagia is probably a result of the increased weight loss seen. There is also occurring a depletion of the fat stores of the body and will be considered later. The R. Q. decreases because less glucose is being oxidized at the exsiting blood sugar level and more energy is being used for transformative and partially oxidative reactions.[21] The D/N ratio is elevated since more sugar is being eliminated by the kidneys than normally and catabolism of protein has been speeded up.

C. *Glucose Tolerance Test.* — The one hour two dose test of Exton and Rose[69] seems to be the most sensitive and accurate diagnostic test for diabetes mellitus. A dose of dextrose is given and repeated in thirty minutes, while urine and blood samples are being taken. A normal test shows a normal fasting blood sugar level (80-120 mg. per cent), a rise not to exceed 75 mg. per cent in thirty minutes, the same, less, or not greater than 5 mg. per cent rise in sixty minutes over the thirty minute sample, and all urine samples negative to Benedict's test. These results depend on the body's ability normally to convert glucose to glycogen in the liver and the muscles as shown by Soskin et al.[37] Normally, with more glucose added, more glycogen will be formed. Insulin was shown to be necessary in certain amounts for this reaction but did not increase significantly following glucose administration to bring about increased glucose oxidation, as many had previously believed. In a diabetic, with a decreased supply of insulin present, the conversion of glucose $\rightarrow$ glycogen is not as rapid as normal and not as complete at certain blood sugar levels. Therefore, with the Exton Rose test, we see a normal or high fasting level, a rise of greater than 75 mg. per cent in thirty minutes, a rise of not less than 10 mg. per cent in the sixty minute sample over the thirty minute one, and glycosuria in one or more of the urine samples.

D. *Glycosuria.*—Glucose is a "high threshold" substance for the renal mechanism, is normally absorbed with water to a large extent, and consequently is absent from the urine. This reabsorption process is an active one, occurring in the proximal renal tubules as a result of phosphorylation of the glucose molecule by kidney phosphorylase and dephosphorylation by alkaline phosphatase in the tubular cells.

When the blood sugar concentration increases above certain normal limits (about 160 mg. per cent) the active resorption process is slowed or stopped. In addition, the increased concentration of glucose in the glomerular filtrate causes the osmotic pressure at which the reabsorption process ceases to be reached sooner than in the normal kidney.[3] In the loop of Henle this concentration of glucose exerts an osmotic pressure and thereby brings water back into the tubules, increasing the amount of water lost by the body and producing a diuresis.[70] Johns[35], by means of comparing simultaneously drawn blood and urine samples, noted that glycosuria occurred at higher levels on the ascent of the blood tolerance curve than on the descent. Therefore, once the penetration of sugar through the kidney occurs, it continues though a much lower level of blood sugar concentration is present. As a result of the increased loss of sugar with water, polyuria occurs, followed by a polydipsia secondary to the dehydration that occurs. The pruritus seen in women is thought to be secondary to the irritating effect of the increased amounts of glucose in the urine.

E. *Ketosis.*—Knoop, quoted by Stadie,[25] first thought fatty acids to be oxidized by beta oxidation in which two carbon atoms were oxidized at one time with the formation of acetic acid. This continued on the long chain fatty acids until only one ketone body was left, and therefore, many acetic acid molecules were formed with only 1 ketone body. The latter was slowly oxidized only when glucose was simultaneously oxidized. If the glucose was unable to be oxidized or not present, no oxidation of the fatty acid ketone body took place and an accumulation of ketone bodies resulted. This was the "Fats burn in the flame of the carbohydrates" theory of Shaffer.[23] Hurtley in 1916 introduced the multiple alternate oxidation theory saying that oxidation of fatty acids took place at alternate carbon atoms and all at once so that many ketone bodies were produced. Stadie[25] found this to be true by quantitative experimentation and therefore opposed the Knoop theory.

He showed that fatty acids were oxidized by the peripheral tissues with or without insulin or glucose present. He further showed that diabetic tissues "call out" for energy from fat and the liver responds by utilizing fatty acids with the production of ketone bodies. These ketone bodies are utilized by the peripheral tissues. However, if the liver produces them faster than the peripheral tissues can oxidize them, then an excess occurs in the blood stream and in the urine. Stadie[25] stated the following hypothesis: "Up to a certain level all fat catabolized is completely utilized; hence, there is no ketonuria. Beyond this level all fat catabolized is not completely utilized; hence, part of the fat catabolized is excreted in the form of ketone bodies . . . The greater the carbohydrate utilized, the less the fat metabolism; that is, carbohydrate spares fats."

Soskin[21] found, by perfusion experiments, that 10-40 times as many ketone bodies were produced by the liver than by any other organ or tissue. In addition, any hepatotoxic agent decreased the production of ketone bodies, so the liver is definitely the most important organ concerned with ketogenesis. The regulation of the ketone body production will therefore depend on the integrity of the liver cell. In cases of starvation, fatty livers, and so on, there is a tendency to increase ketone production more than in the normal. Soskin mentions the endocrine involvements in the metabolic process. He says that insulin is anti-ketogenic while the thyroid, anterior pituitary, adrenal medulla, and adrenal cortical factors are all ketogenic.

Somogyi and Weichselbaum[72] found ketonuria to increase following glucose administration. If "Fats burn in the fire of carbohydrates", the ketonuria should have decreased so they concluded that carbohydrates do not exert ketolytic effects but exerted ketone sparing effects in various extrahepatic tissues and decreased ketone body utilization.

Barach[73] concluded that ketones make their appearance when inadequate carbohydrate is being metabolized and excess fat is being utilized. So, whether in health or disease, the cause for the ketosis is the same.

F. *Acidosis.*—Soskin and Levine[74] are not at all sure that the increased ketone bodies in the blood are harmful per se. But, as Gamble[75] points out, these ketone acids must be given sodium ions for neutralization purposes in order to retain a normal pH. This sodium ion is given up at the expense of the bicarbonate ion. Therefore, an increase in ketone acids is associated with a reduction of the bicarbonate content of the plasma. In addition, the rapid dehydration almost always occurring in diabetic coma is responsible for certain factors taking place which prevent renal function in the normal manner. As a result of this, more organic acid radicals are retained by the kidney, combine with sodium, and displace more bicarbonate to a very low value. Therefore, acidosis is seen to occur on an easily explainable physiological basis. Whether or not the symptoms of diabetic coma depend upon the acidosis cannot be definitely said. The coma is probably a result of the hemoconcentration of the blood stream with decreased oxygenation to the central nervous system. Soskin and Levine[74] found only evidence of cerebral capillary dilatation, perivascular edema, and acute degenerative changes in the cells of the central nervous system. They explained the changes on the basis of dehydration, hemoconcentration, and cerebral anemia.

CONCLUSION

In conculsion, I would like to repeat that diabetes mellitus is a disorder in carbohydrate metabolism secondary to insulin deficiency. All of the many manifestations of this disease may be explained on that basis. There occurs firstly a reduced rate of carbohydrate oxidation by the peripheral tissues, with a secondary, compensatory over-production of glucose from the liver which initiates the latter changes, ending in ketosis and acidosis. The importance of the liver in the normal and abnormal metabolic processes has been stressed. Therapy should be directed towards replacement of the insulin the body is lacking and replenishing the glycogen depleted liver with a new supply of glycogen in an attempt to restore the normal physiologic functions.

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INFANT MORTALITY IN LOUISIANA AND UNITED STATES

The infant mortality rate per 1,000 live births was 43.0 in Louisiana in 1945 as contrasted with 46.3 in 1944. The drop in infant mortality was in line with that which has occurred throughout most of the states in the Union. The infant mortality rate in Louisiana, however, is higher than that throughout the United States as a whole, which rate is 38.3. Our rate is higher than that in the nearby Southern states: In Mississippi it is 40.6; in Alabama it is 38.3; in Arkansas 31.7. However, in Texas the rate is 48.8 which the Texans probably attribute to the high rate amongst their Mexican population. Incidentally, the state of New Mexico has the highest rate in the Union, nearly 100.8. The lowest rate in the country is in the state of Rhode Island where the mortality rate per 1,000 live births is only 28.2.

While the figure for the maternal mortality rate of each state are not given in this particular bulletin of the U. S. Public Health Service, the statement is made that the rate amongst mothers has diminished nine per cent from 1945.

The diminution in the number of infant and maternal deaths is attributed in part to the large portion of babies who are now delivered in hospitals. In 1945 the figures show that nearly 80 per cent of all deliveries were reported to have occurred in hospitals.

Comment was made concerning the infant death rate among Mexicans. It should also be noted that the infant mortality rates in the South are somewhat higher than in the North and in the West but these rates are always higher in the negro group than they are in the white population.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly this year was a truly tremendous success. The program appealed to doctors from all over the country so that actually checks came from every state in the Union asking that the writer of the check be registered for the Assembly. The unfortunate thing about this meeting was the fact that it was impossible to obtain hotel accommodations for all those who wished to attend the meeting. As a matter of fact several hundred checks had to be returned to the senders because no rooms were to be had for them in the City of New Orleans.

The scientific program was most successful. The out of town speakers for the most part presented their subject matter clearly, succinctly and in a manner that would appeal to and impress itself upon the memory of the physician. Certainly the outside of state speakers could not have been better selected than they were. To Dr. W. H. Gillentine, president of the organization, goes much praise as he has worked assiduously in the interests of the Assembly. We feel sure that Dr. Joseph D'Antoni, the next president, will do equally well and that Dr. Waldemar R. Metz, the president-elect, will ably maintain the standard set by this organization since it has been in existence.

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POSTERIOR PITUITARY EXTRACT TO MEASURE RENAL FUNCTION

Several years ago Sodeman and Engelhardt[1] devised a test for renal function which has many advantages over those that are commonly employed. Undoubtedly the most satisfactory test for renal function in most instances depends upon measuring the concentrating capactiy of the tubules. The disadvantages of previous tests have been the discomfort to the patient because of a long period of water restriction and the necessity of collecting the urine at various times during the period in which the test is going on. Sodeman and Engelhardt found that if they gave posterior pituitary extract to their patients that the results are comparable to those in which the more elaborate procedures are carried out. The only procedure necessary is to give 0.5 c.c. posterior pituitary extract subcutaneously and then collect the urine at one-half hour intervals for two hours. A recent publication by Horne and Morris[2] substantiates what Sodeman and Engelhardt observed in their original studies. Attention is called to this kidney function test and to this particular article, which is one of several that have confirmed the results obtained by the two original authors because the test is simple and dependable and permits, according to Horne and Morris, of a rapid evaluation of the concentrating powers of the kidney. The Bowman Gray School of Medicine observers furthermore found that the test in 25 patients who did not have kidney disease gave results comparable to the fluid restriction test. There was some difference in 27 patients with impaired kidney function but these changes were in no way of special significance because they were based upon the invalid assumption that one can expect the fluid restriction test always to give a constant result. "The pituitrin test determined accurately whether or not a given patient should be classified as having impaired renal function. It may be substituted for the fluid restriction test to measure the concentrating ability of the kidneys . . . ." Horne and Morris noted furthermore that this test accurately estimates the concentrating power of the kidney in patients with edema. Lastly, they concluded that the posterior pituitary test is of considerable value to the busy physician because it can be carried out in his office without any special preparation of the patient, it is finished within two hours and in the average patient causes no disturbing reaction. However, it should be borne in mind that pituitrin should not be given to the pregnant girl and potentially is dangerous to the patient who may have coronary arterial disease. The side effects of pituitrin, when given in doses of 10 U.S.P. units, are very slight. Occasionally the patient has abdominal cramps and in about 40 per cent they may have a painless stool. In no one of the patients studied by Horne and Morris were there any significant changes in the blood pressure. Parenthetically it might be observed that these authors used 1 c.c. of pituitrin and necessarily used a small calibrated urinometer.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

AGREEMENT WITH THE VETERANS ADMINISTRATION

Following is copy of contract and fee schedule now in effect between the Veterans' Administration and the Louisiana State Medical Society for medical care of veterans with service connected disabilities. Cooperation of our members in this project is earnestly requested.

CONTRACT

1. It is the purpose of the Louisiana State Medical Society to collaborate with the Veterans' Administration in a manner which will provide the best possible medical care for veterans residing in the State of Louisiana.

2. The Louisiana State Medical Society will request all of its members to participate in a statewide program whereby physicians in private practice will render medical services (examinations, treatments and counsel) in such cases as may be authorized by the Veterans' Administration.

3. The Louisiana State Medical Society will submit to the Veterans' Administration a list of members who desire to provide services for eligible veterans in home communities of such veterans.

4. The physicians so listed may be appointed as fee-designated physicians of the Veterans' Administration.

5. Such list may be augumented from time to time as additional physicians indicate a desire to participate in the program.

6. By notice in writing, a physician may at any time request that his name shall be removed from the list of fee-designated physicians.

7. Fees for medical services in authorized cases shall be paid by the Veterans' Administration to the physician rendering the service in accordance with the fee schedule, which is attached hereto and made a part of this agreement. The Louisiana State Medical Society warrants that the rates charged herein are not in excess of the rates charged other persons who are not Veterans' Administration beneficiaries for the same service. It is understood that unusually involved cases and services not scheduled will be subject to review by the Louisiana State Medical Society for recommendation to the Veterans' Administration as to the appropriate fee.

8. The Louisiana State Medical Society will assist the Veterans' Administration in establishing for examinations and treatments a list of competent specialists who meet the qualifications for spscialists of the Veterans' Administration.

9. Lists submitted by the Louisiana State Medical Society will be broken down by parishes or districts in order that the veteran for whom services are authorized may select a physician practicing in his home community.

10. The Veterans' Administration will handle administrative and clerical details in connection with the authorization of examinations or treatments and the maintenance or records; and will arrange for transportation of the veteran if necessary.

11. When authorizing examination or treatment, the Veterans' Administration will furnish to the veteran proof of such authorization and a list of fee-designated physicians in the parish or district in which the veteran is located in order that he may select his own physician for the services authorized.

12. The Veterans' Administration will review reports of examinations and services to determine their adequacy. No fees will be paid by the Veterans' Administration for reports which are not acceptable to the Veterans' Administration nor for services rendered in unauthorized cases.

13. The Louisiana State Medical Society will establish one or more boards of review composed of physicians. It shall be the duty of such board to review reports, which are deemed by the Veterans' Administration to be inadequate or which do not meet the requirements of the Veterans' Administration; to recommend, at its discretion, the disqualification of any physician from further work with the Veterans' Administration whose work is found by the board to be incomplete or unsatisfactory; to advise and assist the Veterans' Administration on other matters within the scope of this program.

14. The Louisiana State Medical Society does not propose to make any charge for any service rendered to the Veterans' Administration under this agreement.

15. This contract may be discontinued upon 30 days' notice by either participant.

Signed on behalf of the
Louisiana State Medical Society by:

RHETT McMAHON, M. D.,
President

EDWIN L. ZANDER, M. D.,
Chairman of Council

P. T. TALBOT, M. D.,
Secretary-Treasurer

Signed on behalf of the Veterans' Administration by:

R. C. KIDD, Director, Supply Service
Veterans Administration
Washington 25, D. C.

Effective date of this agreement:
December 1, 1946

FEE SCHEDULE

1. Bronchoscopy	$40.00
2. Bronchoscopy and biopsy	50.00
3. Dermatological examination	7.00
4. Electrocardiogram with interpretation	10.00
5. Esophagoscopy	40.00
6. Examination of ears, nose and throat (separately or together)	5.00
7. Special ear examination, including audiometric test, with chart	10.00
8. Special ear examination to include either caloric or Bárány test, or both, with report	10.00
9. Examination of eyes (to include either a copy of the prescription ordered or the retinoscopic correction of the refractive error, the fundus and field findings—the latter by chart in all cases of optic atrophy)	10.00
10. Examination of eyes with refraction, if mydriatic is used (to include either a copy of the prescription ordered or the retinoscopic correction of the refractive error, the fundus and field findings—the latter by chart in all cases of optic atrophy)	12.50
11. Combined examination of eyes, ears, nose and throat, with refraction (with or without mydriatic)	15.00
12. Gastroscopy	40.00
13. Genitourinary examination without cystoscopy	5.00
14. Genitourinary examination with cystoscopy	15.00
15. Genitourinary examination with cystoscopy and ureteral catheterization	25.00
16. Gynecological examination	5.00
17. Complete examination of heart, including electrocardiography	15.00
18. Physical examination of heart or lungs, or both	10.00
19. Neurological examination (complete)	5.00
20. Neuropsychiatric examination (complete)	10.00
21. Routine office examination, including treatment	3.00
22. Orthopedic examination	7.50
23. Physical examination to determine need for hospitilization	3.00
24. Complete physical examination	7.50
25. Proctoscopy or sigmoidoscopy	7.50
26. General surgical examination	5.00

SIMPLE FRACTURES

27. Carpal bone, one	35.00
28. Carpal bones, each additional	5.00
29. Clavicle	40.00
30. Coccyx removal	50.00
31. Femur	125.00
32. Femur, when suture, plating or nailing is necessary	135.00
33. Fibula or tibia, or both (including Pott's fracture)	75.00
34. Fibula or tibia, or both (including Potts fracture) when suture or plating is necessary	100.00
35. Finger, one	15.00
36. Fingers, each additional	5.00
37. Humerus	75.00
38. Humerus, when suture or plating is necessary	100.00
39. Malar bone	20.00
40. Maxilla inferior (wiring if necessary)	75.00
Double	100.00
41. Maxilla superior (wiring if necessary) or raising	75.00
42. Metacarpal bone, one	15.00
43. Metacarpal bones, each additional	5.00
44. Metatarsal bone, one	20.00
45. Metatarsal bones, each additional	5.00
46. Nasal bones	25.00
47. Patella	50.00
48. Patella, when suture or plating is necessary	75.00
49. Pelvis	100.00
50. Pelvis, when suture or plating is necessary	125.00
51. Radius or ulna, or both (including Colles' fracture)	50.00
52. Radius or ulna, or both (including Colles' fracture) when suture or plating is necessary	75.00
53. Rib, one	15.00
54. Ribs, each additional	5.00
55. Sacrum	50.00
56. Scapula	50.00
57. Skull	100.00

58. Sternum	75.00
59. Tarsal bone, one	15.00
60. Tarsal bones, each additional	5.00
61. Toe, one	15.00
62. Toes, each additional	5.00
63. Vertebra, one or more	100.00

Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

COMPOUND FRACTURES

In all compound fractures add 40 per cent additional fee.

DISLOCATIONS

64. Carpal bone, one	25.00
65. Carpal bones, each additional	5.00
66. Clavicle	35.00
67. Elbow	35.00
68. Finger, one	10.00
69. Fingers, each additional	5.00
70. Hip	60.00
71. Knee	50.00
72. Maxilla, inferior	15.00
73. Metacarpal bone, one	15.00
74. Metacarpal bones, each additional	5.00
75. Metatarsal bone, one	15.00
76. Metatarsal bones, each additional	5.00
77. Patella	40.00
78. Pelvis	75.00
79. Shoulder	40.00
80. Shoulder, recurrent or habitual	35.00
81. Tarsal bone, one	15.00
82. Tarsal bones, each additional	5.00
83. Thumb	15.00
84. Toe, one	10.00
85. Toes, each additional	5.00
86. Vertebra, one or more	100.00

AMPUTATIONS

87. Upper arm	100.00
88. Forearm	75.00
89. Finger, one	25.00
90. Fingers, each additional	10.00
91. Foot	75.00
92. Hand	75.00
93. Leg	75.00
94. Thigh	100.00
95. Toe	25.00
96. Toes, each additional	10.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

ABSCESS, INCISION AND DRAINAGE

97. Brain abscess	150.00
98. Cellulitis, incision and drainage	25.00
99. Deep abscess (including ischiorectal)	40.00
100. Empyema, incision and drainage, including rib resection	100.00
101. Liver abscess	150.00
102. Oral abscess (not to include dental or peridental)	15.00
103. Prostatic abscess, incision and drainage	75.00
104. Superficial abscess	5.00
105. Subphrenic abscess	150.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees except as to items numbered 99 and 104.

OPERATIONS

106. Abdominal fixation for prolapse of rectum	125.00
107. Adenectomy, cervical, inguinal, etc. (minor)	20.00
108. Adenectomy, cervical, inquinal, etc. (radical)	125.00
109. Anal fissure, operation for	75.00
110. Anastomosis, intestinal	150.00
111. Anastomosis, uretero-intestinal	150.00
112. Ankle joint, excision of	75.00
113. Apicolysis	100.00
114. Appendectomy	100.00
115. Arthroplasty, major joint	125.00
116. Biopsy	10.00
117. Bone graft (long bones)	150.00
118. Bone plate, removal of	50.00
119. Breast, resection of (simple)	75.00
120. Breast, resection of (radical)	150.00
121. Carcinoma of lower lip, excision of	50.00
122. Carcinoma of rectum, excision of	200.00
123. Carcinoma of tongue, excision of	75.00
124. Cardiospasm, dilatation for	25.00
125. Cartilage of condyle of femur, removal of	70.00
120. Breast, resection of (radical)	150.00
joint	75.00
127. Cervix, amputation of	75.00
128. Cholecystectomy	150.00
129. Cholescystotomy	100.00
130. Choledochotomy	150.00
131. Chordotomy	125.00
132. Circumcision	25.00
133. Clawfoot, operation for	75.00
134. Coccyx, excision of	50.00
135. Colostomy	100.00
136. Colporrhaphy and perineorrhaphy	100.00
137. Cystotomy, suprapubic	75.00
138. Dupuytren's contraction, operation for	100.00
139. Elbow joint, excision of	75.00
140. Epididymectomy	40.00
141. Esophagus, dilatation of by means of bougies or sounds	10.00
142. Femoral artery, ligation of	75.00
143. Fecal fistula, abdominal, operation for	150.00
144. Fistula, rectovaginal, operation for	100.00

145. Fistula, urethral, operation for	
First	50.00
Second	25.00
146. Fistula, vesicovaginal, operation for	75.00
147. Fistula-in-ano, operation for	75.00
148. Fulguration of tumor of bladder	
Closed	50.00
Open	75.00
149. Fulguration of tumor, superficial	10.00
150. Gasserian ganglion, excision of	150.00
151. Gastrectomy (partial)	150.00
152. Gastroenterostomy	150.00
153. Hallux valgus, operation for	75.00
154. Hallux valgus, bilateral, operation for	75.00
155. Hammer toe, operation for	25.00
156. Heart, operation on	150.00
157. Hemorrhoidectomy	75.00
158. Herniotomy, diaphragmatic	150.00
159. Herniotomy, ventral, $100.00, inguinal, $75.00, or femoral	75.00
160. Herniotomy, ventral, $100.00, inguinal, or femoral (bilateral)	125.00
161. Hip joint, excision of	150.00
162. Hydrocele, aspiration of	5.00
163. Hydrocele, operation for	50.00
164. Hysterectomy, abdominal or vaginal (including removal of adnexa, if indicated)	150.00
165. Ingrown toenail, excision of	15.00
166. Intestinal obstruction, operation for	100.00
167. Knee joint, excision of	100.00
168. Laminectomy	150.00
169. Laparotomy and drainage, general peritonitis	125.00
170. Litholapaxy	75.00
171. Lobectomy	150.00
172. Meckel's diverticulum, excision of	125.00
dependent on site	$40.00—100.00
173. Nephrectomy or nephrotomy	150.00
174. Nephropexy	150.00
175. Nerve, suture of	100.00
176. Supraorbital nerve, injection of	10.00
177. Neuroma, resection of	75.00
178. Orchidectomy	75.00
179. Osteomyelitis, operation for—dependent on site	$40.00—100.00
180. Ovariotomy	100.00
181. Papilloma of bladder, operation for	100.00
182. Paracentesis of abdomen	15.00
183. Paracentesis of pericardium	25.00
184. Paracentesis of thorax	25.00
185. Perineum, repair of	75.00
186. Phrenic nerve operation	75.00
187. Pneumolysis, extrapleural or intrapleural	100.00
188. Pneumonectomy	200.00
189. Pneumonotomy, cautery	100.00
190. Pneumoperitoneum, first induction	25.00
191. Pneumoperitoneum, refills	10.00
192. Artificial pneumothorax, first induction	25.00
193. Artificial pneumothorax, refills	10.00
194. Prostatectomy, perineal	150.00
195. Prostatectomy, suprapubic (one or two stages)	150.00
196. Prostatic resection, transurethal	125.00
197. Cisterna puncture, including local anesthetic and obtaining fluid	50.00
198. Lumbar puncture, including local anesthetic and obtaining fluid	10.00
199. Pyelotomy, with removal of calculus	150.00
200. Pyloroplasty	125.00
201. Salpingectomy	150.00
202. Scaleniotomy	50.00
203. Sequestrum, removal of (deep) depending on size and location	150.00
204. Sequestrum, removal of (superficial)	25.00
205. Shoulder joint, excision of	125.00
206. Skull, decompression of	150.00
207. Fixation of spine, operation for (Albee or Hibbs')	150.00
208. Splenectomy	125.00
209. Stricture of rectum, operation for	50.00
210. Sympathectomy, cervical	175.00
211. Sympathectomy, periarterial	100.00
212. Tenorrhaphy, one	40.00
213. Tenorrhaphy, each additional	15.00
214. Tenotomy	35.00
215. Thoracotomy	100.00
216. Thoracoplasty, each stage	125.00
217. Thyroid artery, ligation of	75.00
218. Thyroidectomy	150.00
219. Torticollis, operation for	75.00
220. Tumor, abdominal, removal of	125.00
221. Tumor of brain, operation for	200.00
222. Tumor, gastrointestinal tract, resection of, including intestinal anastomosis	200.00
223. Tumor or cyst, removal of, depending on site	$25.00—50.00
224. Tumor or cyst, removal of	10.00
225. Ulcer, gastric or duodenal, operation for	150.00
226. Ureteral stone, removal of	125.00
227. Urethral stricture, dilatation of	5.00
228. Urethrotomy, external	40.00
229. Urethrotomy, internal	40.00
230. Prolapsus uteri, operation for, including perineal repair	150.00
231. Uterine displacement, abdominal, operation for	125.00
232. Uterus, dilatation and curretage of	50.00
233. Varicocele, operation for	50.00
234. Varicose veins, injection treatment, each injection	5.00
235. Varicose veins, one leg, operation for	75.00
236. Varicose veins, both legs, operation for	125.00
237. Venesection	15.00
238. Whitehead's operation	75.00

239. Wrist joint, excision of	75.00

Note—these amounts include 30 days' postoperative care, exclusive of hospital charges, anesthetic and x-ray fees.

SURGICAL CARE OF TRAUMATIC WOUNDS

240. Incised, depending on size and location	$5.00—50.00
241. Lacerated, depending on size and location	$5.00—50.00
242. Punctured, depending on size and location	$5.00—50.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

ANESTHESIA

243. Avertin anesthesia	10.00
244. General anesthesia	
(a) By visiting physician	15.00
(b) By intern or nurse	5.00
(In case of gas anesthesia, an additional allowance of $5.00 may be authorized for the gas used.)	
245. Local anesthesia—No fee will be allowed for local anesthesia in cases which require a local anesthetic for examination, treatment, or surgical operation.	
246. Rectal anesthesia	10.00
247. Spinal anesthesia, including anesthetic	20.00

EYE OPERATIONS

248. Cataract, needling operation for	50.00
249. Cataract, operation for	100.00
250. Chalazion, operation for	10.00
251. Corneal ulcer, cauterization of	10.00
252. Extensive peripheral ulcer, cauterization of	20.00
253. Ectropion, operation for	50.00
254. Entropion, operation for	50.00
255. Enucleation of eye	75.00
256. Foreign body, removal from conjunctiva (dissection)	15.00
257. Foreign body, removal from conjunctiva (magnet)	10.00
258. Foreign body, removal from cornea (dissection)	25.00
259. Foreign body, removal from cornea (magnet)	20.00
260. Foreign body, removal from eyeball (deep)	$50.00—75.00
261. Grattage of lids for trachoma	10.00
262. Hordeolum, operation for	5.00
263. Iridectomy	75.00
264. Lacrymal duct, dilatation of	10.00
265. Lacrymal sac, excision of	50.00
266. Pterygium, operation for	50.00
267. Ptosis, skin and tarsal resection, operation for	75.00
268. Strabismus, operation for	100.00

Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

EAR OPERATIONS

269. Mastoid, acute, operation for	100.00
270. Mastoid, radical, operation for	150.00
271. Ossiculectomy	75.00
272. Paracentesis	10.00
273. Polypus, removal of	25.00
274. Lateral sinus, drainage of	150.00

Note—these amounts include 30 days' routine after-care exclusive of hospital charges, anesthetic, and x-ray fees.

NOSE AND THROAT OPERATIONS

275. Adenoidectomy	25.00
276. Antrum, intranasal, drainage of	25.00
277. Antrum, radical, operation for	100.00
278. Cleft palate, operation for, complete closure	150.00
279. Harelip, operation for	75.00
280. Intubation	35.00
281. Laryngectomy	150.00
282. Larynx, cauterization of	10.00
283. Tumor of larynx, removal of	100.00
284. Nasal polypus, removal of	25.00
285. Nasal septum, submucous, resection of	75.00
286. Pharyngeal abscess, operation for	20.00
287. Accessory nasal sinuses, irrigation of	10.00
288. Ethmoid sinus, radical, operation for	75.00
289. Frontal sinus, intranasal, drainage of	50.00
290. Frontal sinus, radical, operation for	100.00
291. Sphenoid sinus, drainage of	50.00
292. Tonsillar abscess, operation for	20.00
293. Tonsillectomy	35.00
294. Tonsillectomy and adenoidectomy	45.00
295. Tracheotomy	75.00
296. Turbinate bone, galvano-cauterization of	20.00
297. Turbinectomy	25.00

Note—these amounts include 30 days' routine after-care, exclusive of hospital charges, anesthetic, and x-ray fees.

X-RAY WITH INTERPRETATION

298. Abdomen, flat plate	10.00
299. Ankle joint, anteroposterior and lateral views	7.50
300. Arm, humerus, anteroposterior and lateral views	10.00
301. Bladder, with injection, anteroposterior view	10.00
302. Chest, for pulmonary, cardiac or rib fracture diagnosis, plain	10.00
303. Chest, for pulmonary, cardiac or rib fracture diagnosis, stereoscopic	15.00
304. Clavicle, anteroposterior view	7.50

305. Elbow, anteroposterior and lateral views	7.50
306. Fluoroscopy, when required, without film	5.00
307. Foot, anteroposterior and lateral views	7.50
308. Forearm, radius and ulna, anteroposterior and lateral views	7.50
309. Foreign body in eye, location of (the fragment charted in three planes and its dimensions ascertained by the method of Sweet or equivalent)	25.00
310. Gall bladder, Graham technic, including cost of dye	15.00
311. Gastrointestinal tract, complete x-ray study including fluoroscopy	35.00
312. Hand, anteroposterior and lateral views	5.00
313. Hip joint, anteroposterior view, plain	10.00
314. Hip joint, anteroposterior view, stereoscopic	15.00
315. Intestine, barium clysma, 14 by 17 films for position and outline	10.00
316. Jaw, upper and lower	7.50
317. Kidneys, right and left for comparison	10.00
318. Knee joint, anteroposterior and lateral views	7.50
319. Leg, tibia and fibula, anteroposterior and lateral views	7.50
320. Lipiodol injection for bronchiectasis, etc., including roentgenograms	25.00
321. Pelvis, anteroposterior view, plain	10.00
322, Pelvis, anteroposterior view, stereoscopic	15.00
323. Pyelography, using uroselectan or similar preparation (including cost of drug)	15.00
324. Pyelography, retrograde	25.00
325. Scapula	7.50
326. Shoulder joint, anteroposterior view, plain	7.50
327. Shoulder joint, anteroposterior view, stereoscopic	10.00
328. Sinuses, frontal and ethmoid, anteroposterior and lateral views	10.00
329. Sinuses, mastoid, right and left sides for comparison	10.00
330. Sinuses, maxillary, anteroposterior and lateral views	10.00
331. Sinuses, frontal, ethmoid and maxillary, anteroposterior and lateral views	15.00
332. Skull, anteroposterior and lateral views, plain	10.00
333. Skull, anteroposterior and lateral views, stereoscopic	15.00
334. Spine, cervical, anteroposterior and lateral views	10.00
335. Spine, dorsal, anteroposterior and lateral views	12.50
336. Spine, lumbosacral, with coccyx, anteroposterior and lateral views	12.50
337. Spine, entire	22.50
338. Stomach, barium or bismuth meal, 14 by 17 film; after ingestion, four 8 by 10 films for detection of duodenal cap; total of five films including fluoroscopy	25.00
339. Teeth, single (up to and including 7 films) each	1.00
340. Teeth, series (7 films to and including full mouth)	7.00
341. Thigh, femur, anteroposterior and lateral views	7.50
342. Wrist, anteroposterior and lateral views	5.00

INTERPRETATION OF ROENTGENOGRAMS

343. Bones and joints, plain anteroposterior and lateral views	5.00
344. Chest for pulmonary diagnosis, plain or stereoscopic	5.00
345. Gastrointestinal series	5.00
346. Genitourinary tract	5.00
347. Kidney films	5.00
348. Skull, following ventriculography or encephalography	5.00
349. X-ray therapy, deep, per treatment (Maximum expenditure allowed not to exceed $100.00; where additional treatments are necessary, special authority must be obtained from Central Office.)	$10.00—25.00
350. X-ray therapy, superficial	5.00

MISCELLANEOUS

351. Blood transfusion	35.00
352. Amount allowed for blood furnished, when not donated, per 100 c.c.	5.00
353. Application of plaster cast, chest (including material)	15.00
354. Application of plaster cast, thighs and hips (including material)	25.00
355. Application of plaster cast, thigh and leg (including material)	10.00
356. Application of plaster cast, torso (including material)	25.00
357. Application of plaster cast, torso and hips (including material)	30.00
358. Application of plaster cast, torso, entire body, (chest to feet, including material)	50.00
359. Application of plaster cast for disease or injury of vertebrae (including material)	15.00

Office visit	$2.00
Day calls to home or hospital	3.00

Night calls to home or hospital (For all calls within city limits)	5.00
Mileage (one way within zone, beginning at city limits, per mile)	.75

It will be the duty of the Louisiana Medical Society to see that the state is zoned whereby the nearest physician in rural districts must be called in case of emergency. The veteran calling a more distant physician will pay the additional mileage.

CLINICAL LABORATORY EXAMINATIONS

BACTERIOLOGICAL EXAMINATIONS

1. Cultural examination for fungi	$5.00
2. Microscopic examination for fungi	2.00
3. Pneumococcus typing	5.00
4. Pus or exudate (smear)	1.00
5. Pus or exudate, cultural exam, classification	5.00
6. T. Pallidum (dark field)	5.00
7. Throat culture, classification of organism	5.00
8. Throat smear	1.00

BLOOD

9. Agglutination test for typhoid, paratyphoid, or undulant fever	2.00
10. Bleeding time	1.00
11. Blood calcium	3.00
12. Blood chlorides	3.00
13. Blood culture, including classification	5.00
14. Blood platelet count	1.00
15. Blood smear for malaria	2.00
16. Blood typing (grouping)	1.00
17. Carbon dioxide combining power of blood plasma	5.00
18. Chemical examination of blood, complete, including creatinin, dextrose, urea, nitrogen (or non-protein N) and uric acid	15.00
19. Cholesterol	3.00
20. Coagulation time	1.00
21. Complement fixation test — gonococcus infection	3.00
22. Complement fixation test for syphilis	3.00
23. Complement fixation test for tuberculosis	3.00
24. Creatinin	3.00
25. Dextrose	3.00
26. Total erythrocyte count	2.00
27. Fragility test for erythrocytes	5.00
28. Hemoglobin estimation	2.00
29. Hydrogen ion concentration	5.00
30. Differential leukocyte count	2.00
31. Total leukocyte count	2.00
32. Complete blood count, including total counts	5.00
33. Non-protein nitrogen	3.00
34. Non-protein nitrogen	3.00
35. Blood phosphorus	2.00
36. Precipitation test for syphilis	2.00
37. Reticulocyte count	2.00
38. Sedimentation rate	2.00
39. Estimation of sugar tolerance	10.00
40. Urea nitrogen	3.00
41. Uric acid	3.00
42. Van den Bergh blood test for icterus	2.00
43. Volume index	3.00

FECES

44. Cultural exam. of feces for causative micro-organism (classification of bacterium)	7.50
45. Fat in feces	1.00
46. Parasites and ova	5.00

PATHOLOGICAL EXAMINATIONS

47. Autopsy, complete, with report, including histological examination	50.00
48. Tissue examination, with report	5.00

SKIN TESTS

49. Protein sensitization tests (series) including allergens, for purpose of establishing causative factor	25.00
50. Tuberculin	3.00

SPINAL FLUID

51. Examination of spinal fluid for causative organism (smear)	3.00
52. Cell count	2.00
53. Colloidal gold reaction	5.00
54. Complement fixation test for syphilis	3.00
55. Cultural exam. of spinal fluid, including the classification of causative microorganisms	5.00
56. Globulin test	1.00
57. Complete exam. of spinal fluid, including complement fixation test, colloidal gold, globulin test, and cell count	10.00
58. Precipitation test for syphilis	3.00

SPUTUM

50. Tubercle bacillus (plain smear)	3.00
60. Tubercle bacillus (concentration method)	5.00
61. Exam. of duodenal content for pancreatic ferments	3.00
62. Exam. of gastric content for acidity, by histamine	7.50
63. Exam. of gastric content for pepsin	3.00
64. Routine chemical (including test meal with withdrawal of stomach contents)	10.00

URINE

65. Chemical examination, routine	1.00
66. Chemical and microscopical examination	2.00
67. Chlorides	3.00
68. Creatinin	3.00
69. Cultural exam., including classifica-	

	tion of microorganism	5.00
70.	Hydrogen ion concentration	1.00
71.	Mosenthal test	2.00
72.	Total nitrogen	3.00
73.	Renal function test (including phenosulphonephthalein)	3.00
74.	Tubercle bacilli	3.00
75.	Urea nitrogen	3.00
76.	Uric acid	3.00
77.	Urobilin	1.00

MISCELLANEOUS EXAMINATIONS

78.	Animal inoculation for diagnosis, with report of autopsy	10.00
79.	Preparation of autogenous vaccine	10.00
80.	Determination of basal metabolic rate	5.00

MISCELLANEOUS

81.	Blood transfusion (administration only, without venesection)	5.00
82.	Non-surgical drainage of gallbladder	10.00
83.	Electrocardiograms, interpretation of	5.00
84.	Hypodermoclysis	3.00
85.	Injection of alcohol, trigeminal nerve	25.00
86.	Intravenous injection, exclusive of cost of drug	3.00

LOUISIANA STATE PHARMACEUTICAL ASSOCIATION

The Veterans' Administration-Pharmacy Service Agreement with the Louisiana State Pharmaceutical Association has been approved and is now in effect. Information as to participating member pharmacies may be secured by contacting the Veterans' Advisory Bureau, L.S.P.A., Sta. B.—P. O. Box 5317, New Orleans 15.

There are no restrictions upon physicians as to medical agents which may be prescribed but the term "medical requisite" in the agreement is defined as including only the following items: Insulin syringe and two needles; two hypodermic (insulin type) needles; atomizer; nebulizer; hot water bottle; fountain syringe; combination hot water bottle and syringe; ice bag; ice cap; urinal; bed pan; enema can; feeding tube; ear and ulcer syringe. These items may be prescribed only in the case of an emergency and each prescription must be limited to a single item. Pharmacists can not be reimbursed for refilling any prescription and for this reason it will be necessary for the doctors of the state to write new prescriptions whenever additional drugs are indicated or a new medical requisite is required.

For additional data contact the Veterans' Advisory Bureau, L.S.P.A. at the above mentioned address.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

March 10	Scientific meeting, Orleans Parish Medical Society, 8 p. m.
March 12	Woman's Auxiliary, Orleans Club, 3 p. m. Touro Infirmary Staff, 8 p. m.
March 14	Executive Committee, Hotel Dieu, 8 p. m.
March 17	Hotel Dieu Staff, 8 p. m.
March 18	I. C. R. R. Hospital, 12:30 p. m. Charity Hospital Medical Staff, 8 p. m.
March 19	The Orleans Society of X-ray Technicians, 7:30 p. m. Charity Hospital Surgical Staff, 8 p. m.
March 20	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
March 21	Lakeshore Hospital, 8 p. m.
March 25	Baptist Hospital Staff, 8 p. m.
March 26	French Hospital Staff, 8 p. m.
March 27	DePaul Sanitarium Staff, 8 p. m.
March 28	L. S. Faculty Club, 8 p. m. New Orleans Dispensary for Women and Children Staff, 8 p. m.
April 1	Orleans Parish Radiological Society, 7:30 p. m. Eye, Ear, Nose and Throat Staff, 8 p. m.
April 2	Mercy Hospital Staff, 8 p. m.
April 3	Clinico-pathologic Conference, Touro Infirmary, 12 noon. Executive Committee, Baptist Hospital, 8 p. m.
April 4	Ochsner Clinic Staff, 8 p. m.
April 7	Board of Directors, Orleans Parish Medical Society.
April 9	Woman's Auxiliary, Orleans Club, 3 p. m. Touro Infirmary Staff, 8 p. m.

Every Thursday—Medical Toastmasters, New Orleans Athletic Club, 6:30 p. m.

N. J. Tessitore, M. D., Secretary.

NEWS ITEMS

Dr. Jules Myron Davidson was elected national Vice-Grand Council of Phi Delta Epsilon, National Medical Fraternity, at their recent meeting in York City.

Dr. F. E. Lamothe was installed as Potentate of Jerusalem Temple at ceremonies held Thursday, January 9.

Dr. A. Mattes was installed as chief rabban.

Dr. I. L. Robbins spoke on the life of Maimonides, twelfth century theologist and philosopher, at a meeting of the Tulane History of Medicine Society held January 10.

Dr. Alton Ochsner recently attended a meeting of the subcommittee on thoracic surgery of the National Research Council in Washington.

Dr. Ochsner also recently addressed the American Cancer Society in New York, and the Academy of Medicine in Buffalo.

Dr. Daniel N. Silverman attended the first meeting of the 1947 Jefferson County Medical Society in Birmingham, January 20-22. Dr. Silverman spoke on the Recognition of the Usual and Unusual Types of Dysentery and Their Treatments. While in Birmingham Dr. Silverman also addressed the faculty and student body of the Alabama Medical School.

Dr. George Battalora was recently elected President of the Mercy Hospital Staff.

Dr. Battalora succeeds Dr. E. J. Richard, who was named to the executive committee.

Other officers elected were: Dr. F. F. Boyce, vice-president; Dr. Louis Monte, secretary; Dr. Edmond Mickal, treasurer; and Dr. E. L. Zander, member of the executive committee.

Dr. Edmond Souchon was recently installed as President of the Hotel Dieu staff; he succeeds Dr. Lucien A. Fortier.

Other officers installed were: Dr. O. C. Cassegrain, vice-president; and Dr. J. O. Weilbaecher, Jr., secretary-treasurer.

Dr. H. Randolph Unsworth was recently elected as President of the DePaul Sanitarium staff; he succeeds Dr. C. S. Holbrook.

Other officers elected were: Dr. Lucy Scott Hill, vice-president and Dr. Louis J. Dubos, secretary-treasurer.

SCIENTIFIC SESSIONS

The result of the recent survey, concerning the type of scientific programs the members prefer, is as follows:

One hundred, ninety-one members replied.

40	mixed papers only.
37	symposia only.
21	symposia and mixed papers.
18	specialty only.
17	symposia, and schools, hospitals, etc.
14	symposia, mixed papers, and schools, hospitals, etc.
12	symposia and specialty.
8	specialty, and schools, hospitals, etc.
6	symposia, specialty, and schools, hospitals, etc.
5	mixed papers and schools, hospitals, etc.
4	medical school, hospitals, etc. only.
3	all four types of programs.
2	specialty and mixed papers.
1	mixed papers and hospitals only
1	doesn't matter.
2	contained no signature and were not included in the count.

1 in addition to making a selection of the types of meetings, suggested more social affairs.

The Scientific Essays Committee is, at present, preparing programs for the year, if you have not returned your card indicating the type of programs you prefer, or informing the Committee as to whether you wish to participate in the programs, it is not too late to do so. The Committee, in preparing the programs, will make the schedule very flexible.

CHANGES IN ADDRESS

Dr. Peter Everett, 502 Pere Marquette Bldg.

Dr. Prudence E. Prouet, 1007 Pere Marquette Bldg.

Dr. Curtis W. Caine, U. S. Naval Hospital, Portsmouth, Virginia.

Dr. J. Wm. Rosenthal, U. S. Veterans Hospital, Fayetteville, Arkansas.

Dr. George E. Scheer, U. S. Quarantine Station, Rosebank, Staten Island 5, New York.

CHANGES IN MEMBERSHIP ROLLS

Dr. W. J. Gilbert, an active member, and Dr. Carey C. Womble, an associate member, were elected to inactive membership.

Dr. Paul M. Goldfarb transferred from intern to active membership.

Drs. Milton R. Cohen, Ralph H. Heeren, Joseph D. Lea, and Roland F. Phillips, all active members, were removed from the rolls because of removal from New Orleans.

HOSPITAL NEWS

The following New Orleans hospitals recently received from the American College of Surgeons, certificates of approval for complying satisfactorily with fundamental requirements for caring for the sick and injured:

Charity, DePaul, Eye Ear Nose and Throat, Flint-Goodridge, Hotel Dieu, Illinois Central, Mercy, New Orleans Hospital Dispensary for Women and Children, Southern Baptist and Touro Infirmary.

NEW ORLEANS MEDICAL FOUNDATION

On January 14 the New Orleans Medical Foundation held its first annual meeting.

The secretary, Dr. L. Sidney Charbonnet, Jr., reported on the activities of the year—the main endeavor being the establishment of the Lakeshore Hospital; he reported that 2,500 patients had been treated in the Hospital between the dates of June 1, 1946 and January 1, 1947, the census having averaged 78 patients. Dr. Charbonnet further pointed out the necessity of complete cooperation of the membership to increase the census and thus meet the expenses of operation of the Hospital, which has maintained very high standards of service.

The treasurer, Dr. Pascal Danna, reported that $125,000 had been expended in the renovation, equipping and organization of Lakeshore Hospital, and that the operating cost averaged $35,000 a month.

Dr. W. P. Gardiner, director of the Hospital, stressed the excellent services that are available for both patients and doctors, and invited all the doctors of the Society to utilize the Hospital facilities which now include adequate beds on all services.

Election of officers was carried out at this meeting—the following were re-elected for the coming year: Dr. Charles B. Odom, president; Dr. J. O. Weilbaecher, Jr., first vice-president; Dr. C. Barrett Kennedy, second vice-president; Dr. L. Sidney Charbonnet, Jr., secretary; Dr. Pascal L. Danna, treasurer; to the executive committee (members-at-large), Drs. George M. Haik, Warren L. Rosen, Chester S. Fresh, Gilbert C. Tomskey, and Alfred B. Longacre.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THIRD DISTRICT MEDICAL SOCIETY

At a recent meeting of the Third District Medical Society, reactivated on 15 January, since its inactivation during the war, there was the largest representation proportionately present at any time in the district's history.

Dr. F. H. Davis, of Lafayette, was elected president, Dr. L. A. Morough, vice-president, Drs. L. Villien and H. M. Flory, delegate and alternate, respectively, to the state convention. Dr. O. E. Dalton was elected secretary-treasurer.

Dr. W. P. D. Tilly gave a very comprehensive and interesting dissertation on medical society organization in this area. Dr. Rhett McMahon, president of the Louisiana State Medical Society, gave a brief talk on unity of the medical profession, socialized medicine, and the future of our Louisiana Physicians Service, Inc. It was unfortunate that Dr. McMahon's talk (especially the extemporaneous parts were not reported verbatim). Dr. Talbot was kind enough to stimulate the meeting with his presence. Mr. Frank Lais, the executive secretary of the Louisiana Physicians Service, Inc., gave a brief explanation of what could be expected from it.

Dr. C. C. DeGravelles, of New Iberia, made a motion which was seconded, and carried, that the next meeting be held in New Iberia.

Dr. L. J. Sonnier acted as secretary.

Orien E. Dalton, M. D.,
Secretary-Treasurer.

WEBSTER PARISH MEDICAL SOCIETY

The Webster Parish Medical Society met at 7:00 p. m., January 27, 1947 at the Imperial Hotel in Minden, La. Supper was served to 50 members, guests, and their wives. Dr. Alton Ochsner of New Orleans spoke on Frequent Errors in the Early Diagnosis and Treatment of Cancer.

E. S. Rogers, M. D.,
Secretary.

THE IBERIA PARISH MEDICAL SOCIETY

The Iberia Parish Medical Society met on January 23, 1947 at a supper-meeting in New Iberia.

Definite steps were taken to hold meetings quarterly and adopt a scientific program to be presented by the members, talks of five minutes with two minutes' discussion periods. Presiding was Dr. Leon Slipakoff, president, and Dr. I. W. Gajan, secretary-treasurer. Members present were Drs. Tilly, DeGravelles, Phillips, Dupuy, Ed L. Landry, Flory, and Dalton. Officers elected for 1947 are: President, Dr. O. E. Dalton; Vice-President, Dr. C. W. Phillips; Secretary-Treasurer, Dr. I. W. Gajan, Jr.; Delegate, Dr. E. L. Landry; Alternate, Dr. Leon Slipakoff.

I. W. Gajon, Jr., M. D.
Secretary-Treasurer.

RAPIDES PARISH MEDICAL SOCIETY

The regular meeting of the Rapides Parish Medical Society was held at the Hotel Bentley on February 3. The meeting was preceded by a dinner after which the scientific program was arranged for by the members of the staff of the Veterans Hospital at Alexandria.

TOURO INFIRMARY

The regular meeting of the Medical Staff of Touro Infirmary was held on Wednesday, February 12 at 8 p. m. Miss Ruth Corpron gave a statistical study of parasitology in the hospital. Dr. Michael DeBakey reported on a case of esophagogastrectomy and Dr. J. K. Howles spoke about lymphoblastoma of the skin.

THE WILLIS-KNIGHTON CLINIC

The Willis-Knighton Clinic announces the following additions to its staff: Dr. Frank L. Bryant, formerly of Minneapolis, who was chief of ophthalmology and otolaryngology in the Oak Ridge, Tennessee, Hospital during the war. Dr. B. H. Young, who was in the Medical Corps of the U. S. Navy, and who is a graduate of Tulane University, has become affiliated with the department of internal medicine. Dr. Herbert H. Vaughan, Jr., formerly associated with the hospital and instructor in the University of Michigan School of Medicine, is now connected with the department of surgery.

CONGRESS ON OBSTETRICS AND GYNECOLOGY

The third American Congress on Obstetrics and Gynecology will be held in St. Louis, September 8-12, 1947. Dr. Woodard Beacham is on the committee in charge of the technical exhibits.

Dr. Fred L. Adair is General Chairman of the Congress. Papers will be presented by leading obstetricians, gynecologists, public health officials, hospital administrators and others. There will be round table discussions and group meetings as well.

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER

A most elaborate and excellent program has been arranged for the meeting of the American Association for the Study of Goiter which will be held at the Biltmore Hotel, Atlantic City, April 2-5, 1947.

THE AMERICAN SOCIETY FOR THE STUDY OF STERILITY

The third annual convention of this organization will be held at the Hotel Strand, Atlantic City, June 7-8, immediately preceding the A. M. A. Convention.

SOUTHERN REGIONAL CONFERENCE ON MEDICAL SERVICE AND PUBLIC RELATIONS

The Council on Medical Service of the American Medical Association has arranged a Southern Regional Conference in cooperation with the Council on Medical Service and Public Relations of the Louisiana State Medical Society. The conference will be held in New Orleans on Saturday, March 15, in the University Room, Roosevelt Hotel. The conference is a one day meeting and registration will start at 9 a. m.

The States of Alabama, Arkansas, Louisiana, Mississippi, Oklahoma and Texas will participate in the conference.

The conference is open to all members of the State Society and members expecting to attend the meeting are advised to communicate with the office advising them of their attendance.

Dr. A. V. Friedrichs is chairman of the arrangements.

Numerous prominent national figures will appear on the program.

9:00 a. m.	Registration.
9:30 a. m.	Word of Welcome—G. C. Anderson, M. D. and H. Ashton Thomas, M. D.
9:45 a. m.	Purposes of Meeting—James R. McVay, M. D.
10:00 a. m.	Liaison between A. M. A. and the State Medical Societies—George F. Lull, M. D., Secretary and General Manager, American Medical Association.
10:30 a. m.	The Washington Legislative Picture —Joseph S. Lawrence, M. D., Director, Washington Office of the Council on Medical Service.
11:00 a. m.	Rural Medical Care—J. Paul Jones, M. D., Camden, Ala. Reports of the States.

12:15 a. m. Luncheon

2:00 p. m. Public Relations Roundtable Discussion—Moderator — Mr. Henry S. Johnson, Director of Public Relations, Medical Society of Virginia.

NEWS ITEMS

The Stars and Bars Chapter of Alpha Omega Alpha held its annual banquet at the St. Charles Hotel on Thursday, February 20. Dr. Dilworth W. Woolley of the Rockefeller Institute for Medical Research addressed the group. On Friday, February 21 the annual oration was delivered by Dr. Woolley in the Hutchinson Memorial on the topic "Development and Uses of Antivitamins."

Dr. George E. Burch, Associate Professor of Medicine at Tulane University, delivered the annual Alpha Omega Alpha address to the members of the chapter at Emory University on February 20. The title of his address was "Studies with Radiosodium in Congestive Heart Failure."

Drs. Edgar Hull, Edward deS. Matthews and Wm. H. Gillentine attended the meeting of the Southeastern Clinical Club in Birmingham on February 15 at which time Dr. Gillentine was elected president of the organization.

Dr. Wm. H. Bloch, of New Orleans, has been honored with life membership in the Alumni Association of the University of Maryland, located at Baltimore. He has been an active member of this association for fifty years.

Colonel Joseph H. McNinch, M. C., has been designated Commandant of the Army Medical Library by The Surgeon General, succeeding Colonel Leon L. Gardner.

Major Douglas Lindsey, who obtained his Majority on October 4, 1946, is at present Deputy Director of the Department of Tactics and Technique at the Medical Field Service School, Fort Sam Houston, Texas.

The American Association of Obstetricians, Gynecologists and Abdominal Surgeons announces a foundation prize contest.

For further information write Dr. James R. Bloss, Secretary, 418 Eleventh Street, Huntington 1, West Virginia.

DOCTOR WANTED

Rating Specialist, Medical, is needed by the Shreveport Regional Office of the Veterans Administration. For details contact office of the State Society or Mr. Thomas Hurt, Shreveport Regional Office.

PHYSICIANS IN CIVILIAN PRACTICE DURING WAR URGED TO REPLY ON NEW QUESTIONNAIRE

Parallel with the advance of scientific warfare there has been a rapid advance in scientific medicine in the twentieth century. The huge strides made in our ability to destroy life and health in the recent war, however, have left many problems which remain to be solved. One of the most critical problems, as recognized by the House of Delegates of the American Medical Association in its December meeting is that of providing care for the civilian population in the event of another national emergency.

The medical profession is wisely assuming its obligation to the American people to be prepared with facts and recommendations for any such future emergency. As a first step in this preparation, 5,000 questionnaires have gone out to a list of physicians, selected at random, who passed the recent war years in civilian practice.

Every physician receiving one of these questionnaires has a professional and humanitarian duty to perform in completing it and returning it promptly. It is preferred that each sign his name, but if anyone wishes to remain anonymous he may do so. The important thing is to get as broad a representation of facts, experiences, and opinions as possible.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana in the week ending January 11 showed the following diseases appearing in numbers greater than ten: 58 cases of pulmonary tuberculosis, 26 of influenza, 20 of septic sore throat and 14 of typhus fever. There were two cases of poliomyelitis listed this week. Of the 14 cases of typhus fever, seven of them were reported from Orleans Parish.

For the week which closed January 18, the indications are that there was a widespread epidemic of mumps as 335 cases were reported this week, followed in order of frequency by chickenpox in 69 instances, 35 of influenza, and 25 of pulmonary tuberculosis. There were no cases of poliomyelitis reported this particular week.

In the week ending January 25, pulmonary tuberculosis led all other reportable diseases with 32 cases, followed by 29 of influenza and 14 of chickenpox. Two cases of poliomyelitis were listed this week and two of malaria.

MONTHLY MORBIDITY FOR VENEREAL DISEASES, STATE OF LOUISIANA

Month Ending December 31, 1946

	Total This Month	Total Previous Months	Total to Date 1946
Chancroid	29	595	624

Gonorrhea	1,034	16,995	18,029
Granuloma inguinale	20	280	300
Lymphopathia venereum	7	99	106
Syphilis	900	18,250	19,150

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending January 4 there were 159 deaths in the City as contrasted with 172 the previous week. These deaths were divided 104 white, 55 negro and of the group 22 were children under one year of age.

For the following week which closed January 11 almost exactly the same number of people died in the city as had died the previous week, namely 156. Of these 100 were white, 56 were non-white and 12 were children under one year of age.

The following week which terminated January 18, showed a remarkable reduction in the number of deaths in the city, in that 113 of the citizens of the city expired this particular week. Seventy-four of these were white, 39 were negro and only 9 were children under one year of age.

The following week which ended January 25, as would be expected, showed a sharp increase in the number of deaths in the city. This week there were 165 deaths divided 108 white, 57 negro and seven infants.

There have been marked fluctuations in the deaths in the city. In the week which began the month of February there was a drop of 124 in the number of deaths, divided 76 white, 33 colored, plus 15 infant deaths.

WOMAN'S AUXILIARY

Mrs. Rhodes J. Spedale, public relations chairman of the Woman's Auxiliary to the Louisiana State Medical society announces the beginning of a series of radio health broadcasts entitled "Melody of Life" arranged through the co-operation of radio station WLCS of Baton Rouge.

The first broadcast was scheduled to go on the air at 4 p. m. Tuesday, January 21 and the program will be heard at that same time weekly for 13 weeks.

The broadcasts will be from transcription prepared by the American Medical Association, with the recording and production facilities available in Chicago, where many of the well-known network radio broadcasts originate. Materials are from the library and the publications of the American Medical Association. Numerous eminent physicians, both in medical practice and public health work, have been brought before the microphone to be interviewed for these "platters". All scripts are professionally prepared by experienced radio writers, and the voices, other than those of the doctors, are those of established radio personalities, among whom are included Harriet Hester, formerly of the staff of WLS, Chicago, and June Merrill women's feature announcer for WJJD, Chicago. Harriet Hester and William J. Murphy, veteran writer of medical scripts, have written the continuities.

The series is under the general direction of Dr. W. W. Bauer, director of the Bureau of Health Education of the American Medical Association, who appears on many of the programs, and his associate in the bureau, Dr. William W. Bolton, whose voice also is heard. Dr. Bauer is general director of the radio work of the American Medical association.

Included in these transcriptions will be the lives of the following men: Mendelssohn, Handel, Schumann, Beethoven, Chopin, Mozart, Brahms, Foster, Moussorgsky, Tchaikowski, Dvorak, Borodin and Bach.

ST. TAMMANY PARISH AUXILIARY

Mrs. Arthur D. Long, of Baton Rouge, State president of the Women's Auxiliary to the Louisiana State Medical Society, visited the St. Tammany Parish Auxiliary on Wednesday. Mrs. Long had with her her mother, Mrs. Stewart, of El Paso, Texas, and Miss Stevenson of Baton Rouge.

The meeting was held at the home of Dr. and Mrs. Carl Young and was called by Mrs. Young, who is State Counselor for the Sixth District and a Past State President. Mrs. Herbert Cannon, president of the St. Tammany Parish Medical Auxiliary, conducted the meeting. The State president, Mrs. Long, gave an interesting and instructive talk. Attending the meeting from Bogalusa were Mrs. Desporte, Mrs. Fleming and Mrs. Ward, Mrs. John K. Griffith, from Slidell and Mrs. M. C. Wigington of Hammond. Those from Covington attending were Mrs. H. E. Gautreaux, Mrs. B. B. Warren, Mrs. Thomas J. Healy and Mrs. A. L. Saxon. Beautiful camellias from the garden of the hostess decorated the rooms of the home. Following the business session, a delicate plate lunch was served by the hostess. She was assisted by Mrs. A. L. Saxon.

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BOOK REVIEWS

Atlas of Surgical Approaches to Bones and Joints: By Toufick Nicola, M. D., F. A. C. S. New York, Macmillan Company, 1945. Pp. 218 illus. Price $5.00.

The line drawings in this book are of excellent character. In addition to depicting accurately the anatomy involved in each of the more common approaches to the bones and joints, the accompanying text helps the reader to find the important structures in the area.

The only criticism that can be offered is that some of the indications and contraindications for a given incision or approach might have been added. For the student and beginner in the art of surgery this is unfortunate as Dr. Nicola is certainly able to advise in this respect.

This book, however, is highly recommended to all who perform surgical procedures on the extremities as a means of reviewing and refreshing the memory concerning area anatomy.

Fred S. Marshall, M. D.

The Chemistry of Anesthesia: By John Adriani, M. D., Springfield, Illinois. Charles C. Thomas, 1946. Pp. 536. Price $7.00.

The scope of this book is much wider than that which might be suggested by the title. As well as a consideration of the chemistry of substances which are commonly used as general anesthetics, attention is given to related compounds having a depressant action on the central nervous system but which for various reasons are unsuitable for practical application. In addition other groups of drugs of interest to the anesthetist are included such as the barbituric acid compounds, the opium alkaloids, the analeptics and drugs affecting the autonomic nervous system. Agents used in the field of local anesthesia are also covered. One omission is that of the curare alkaloids and compounds with similar action which are of recent interest in the field of general anesthesia.

The book is divided into three parts. Part I: Inorganic Chemistry Related to Anesthesia; Part II: Organic Chemistry Related to Anesthesia; Part III: Biochemistry Related to Anesthesia. Part I includes an excellent review of the gas laws, the chemistry of nitrous oxide, carbon dioxide and the gases of air and also a chapter on the absorption of carbon dioxide in rebreathing appliances. In Part II the organic compounds are considered according to chemical groups such as hydrocarbons, alcohols, aldehydes and ethers proceeding to such special classes as the non-volatile hypnotics, opium alkaloids, local anesthetics, and analeptics. In the section on Biochemistry (Part III) one chapter is devoted to proposed mechanisms of narcosis while others are concerned with the effects of anesthesia on the composition of body fluids, liver function, urine formation and general metabolic processes. An extensive bibliography is appended.

Although the author has prepared this book primarily for the anesthesiologist, due to the breadth of subject matter the biochemist, pharmacologist, and internist will find it a source of valuable information.

Ralph G. Smith, M. D.

Studies on Brazilian Anophelines from the Northeast and Amazon Regions: By O. R. Causey, L. M. Deane, and M. P. Deane. (The American Journal of Hygiene Monographic Series, No. 18.) Baltimore, the Johns Hopkins Press, 1946. viii, 50 p.; 19 pl. Price $1.10.

This small, well-printed volume contains three sections, each with an illustrated key for the identification of anopheline mosquitoes of the northeast and Amazon regions of Brazil, by means of adult female, male genitalic, and larval characteristics, respectively. The first section also presents notes on the malaria vectors, a table for the differentiation of the "tarsimaculatus complex", a table listing 35 species and their geographic distribution in the area, and five plates of line-cut illustrations of wing, leg, palpal, and body characters. The second key is introduced by a short description of male genitalia and of the technic of dissection, includes 34 species, and is illustrated by six half-tone plates, five bearing excellent photomicrographs of diagnostic parts of the genitalia of each species. The key to larvae includes 32 species and is accompanied by descriptions of two larvae (*Anopheles galvaoi* and *A. shannoni*) and a table of the distribution and types of breeding places. Eight plates of line drawings illustrate larval characters.

The authors state that three important malaria vectors—*Anopheles gambiae*, *aquasalis*, and *darlingi*—are among the 35 species of anopheline mosquitoes found in this region during surveys under the auspices of the Ministry of Education and Health of Brazil, the International Health Division of the Rockefeller Foundation and the Institute of Inter-American Affairs. A. *Gambiae* was introduced accidently from Africa and caused severe epidemics of malaria before it was eradicated in 1940. Due to its high susceptibility to malaria, its house-frequenting habits, and its preference for human blood, it is the most dangerous vector known. *A. darlingi* and *aquasalis* are indigenous vectors, the former the more efficient and wherever present always accompanied by malaria. *Darlingi* is dependent upon high humidity and rainfall, occurs chiefly along large river valleys, and has seasonal fluctuations which account for the characteristic small regional epidemics of malaria in the Amazon Valley. *Aquasalis*, a coastal brackish-water species, is important as a vector only when numerous and causes a low rate of endemic malaria rather than severe rainy-season epidemics like *darlingi*. Another common species, *A. albitarsis*, seems to include biologically different subspecies, only one of which appears to play a secondary role in the transmission of malaria as a result of appropriate habits in some localities. *A. pessoai* likewise is a vector only under certain local conditions. These observations again emphasize the complexity of malaria epidemiology and the need for exact biological knowledge and continued research to unravel the tangled web and to guide efficient and intelligent control measures.

This volume should constitute an excellent tool for the working entomologist and malariologist in northern Brazil and adjacent areas. It is a sound contribution to our knowledge of a group of insects that is of outstanding importance in public health.

ALBERT MILLER, Ph. D.

Synopsis of Physiology: By Rolland G. Main, Ph. D. St. Louis, C. V. Mosby Co., 1946. Pp. 341. Price $3.50.

This small volume is the outcome of numerous requests for an up to date review of the most recent advances in physiology. The author assumes that his readers are familiar with the rudiments of the subject and so it is approached from the point of view of a text for advanced students and physicians requiring a quick and authoritative reference. There are no references. The author feels these can be ascertained in standard textbooks. It is well indexed and should serve the purpose for which it is intended.

I. L. ROBBINS, M. D.

Principles of Hematology: By Russell L. Haden, M. A., M. D. Philadelphia, Lea & Febiger, 1946. Pp. 366. Price $5.00.

This is a third edition, thoroughly revised. By this time it needs no introduction to the medical public. Changes in the text necessitated by modern advances in hematology have been noted and incorporated in this new edition, as for example, the technic of bone marrow puncture and the study of bone marrow films. However, the last sentence in the preface to the third edition is somewhat puzzling. Thus to quote: "More complicated methods such as the study of the Rh factor have again been intentionally omitted to keep this volume a simple discussion of the fundamental principles of hematology." All authors, editors, teachers kindly take note. The illustrations are good and the format of the book fine.

I. L. ROBBINS, M. D.

Synopsis of Pathology: By William Arnold Douglas Anderson, M. A., M. D., F. A. C. P., 2nd. ed., St. Louis, The C. V. Mosby Company, 1946. Pp. 741, 327 Illus., 15 color pl. Price $6.50.

In the preface to the first edition (1942), Anderson states that his work is "intended to fill a gap which has existed between the very elementary manuals of pathology and the abundant excellent larger textbooks and reference works." It is felt that such a "gap" did exist and that this "Synopsis" has very adequately and effectively filled it. The present second edition is approximately 100 pages longer than the first and contains 23 more text illustrations. Of the total illustrations 74 are new. There are two less color plates in the second edition. Text material and references have been brought up to date. One is indeed most pleasantly amazed at the conciseness and completeness of so compact a volume. Of course, there are omissions of finer and more technical points, but such omissions are only in accordance with the objective of the book. Special mention must be made of the illustrations. These are abundant and of a most excellent quality. They alone would make this book valuable.

The handy size is also an asset, since it is easily carried. The book would certainly seem to be useful, as suggested by the author, to "the clinician who must maintain familiarity with the foundation sciences of medical practice", as well as to the medical student.

W. H. HARRIS, JR., M. D.

PUBLICATIONS RECEIVED

Grune & Stratton, Inc., New York: Progress In Gynecology by Joe V. Meigs, M. D.

Paul B. Hoeber, Inc., New York: Postgraduate Obstetrics by William F. Mengert, M. D.

Bruce Humphries, Inc., Boston: The Effect of Smallpox on the Destiny of the Amerindian by E. Wagner Stearn, Ph. D. and Allen E. Stearn, Ph. D.

King's Crown Press, Morningside Heights, New York: Motor Disorders in Nervous Diseases by Ernst Herz, M. D. and Tracy J. Putnam, M. D.

Lea & Febiger, Philadelphia: Cushny's Pharmacology and Therapeutics by Arthur Grollman, A. B., Ph. D., M. D., F.A.C.P., and Donald Slaughter, B. S., M. D. The Treatment of Diabetes Mellitus by Elliott P. Joslin, A. M., M. D., Sc. D.

Masson et Cie, Paris, France: L. Tuberculose Renale by Jean Cibert. Le Syndrome de Volkmann by Maurice Cahuzac and Francois Jung.

Oxford University Press, Toronto: Heparin in the Treatment of Thrombosis by J. Erik Jorpes, M. D.

Penguin Books, Inc., New York: The Story of Human Birth by Alan Frank Guttmacher, M. D.

W. B. Saunders Company, Philadelphia: Allergy in Theory and Practice by Robert A. Cooke, M. D., Sc. D., F. A. C. P. Gynecological and Obstetrical Pathology by Emil Novak, A. B., M. D., D. Sc. (Hon. Dublin) F. A. C. S.

Charles C. Thomas, Springfield, Illinois: Office Endocrinology by Robert B. Greenblatt, B. A., M. D., C. M. The Lung by William Snow Miller, M. D., Sc. D.

University of Chicago Press, Chicago: Radical Surgery in Advanced Abdominal Cancer by Alexander Brunschwig, M. D.

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SOCIALIZED MEDICINE*

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State, or socialized, medicine was originated during the 1880's by Bismarck acting on the recommendation of his economic adviser, Adolph Wagner, professor of political science at the University of Berlin. Since then this plan of social insurance has been adopted in over thirty countries, generally as a political, financial and regulatory device. Some countries have restricted coverage while others, like Russia, cover the entire population and carried the compulsory scheme to the point of making all physicians the salaried servants of the State.

Most people, including a large part of the medical profession, associate the beginnings of the agitation for socialized medicine in the United States with advent of the New Deal but actually this is not the fact. According to an article in Medical Economics in November, 1945, entitled "Labor Program to Socialize Medicine Internationally", in this sphere Franklin Delano Roosevelt was no innovator. He was a follower. Plans for socializing medicine throughout the world, one nation at a time, had been drawn up by the International Labor Organization years before. This organization was established in 1919 at Basel, Switzerland, as a function of the League of Nations. In spite of the fact that the United States Senate specifically withheld ratification of the part of the Treaty of Versailles which established the International Labor Organization, nevertheless Franklin D. Roosevelt, while Under-Secretary of the Navy in 1919, made the necessary arrangements for the first international labor conference to be held in the Navy Building in Washington in that year; and in 1934, through a joint resolution of Congress, sponsored by the Roosevelt administration, the United States became a full fledged participating member in the International Labor Organization. This action entitled the United States to official representation on all the International Labor Organization committees of experts. Under this provision, Arthur T. Altmeyer, now Chairman of the Social Security Board, and Wilber Cohen, Assistant Director of Research, soon won places on the International Labor Organization Experts Committee for Medical Care.

A special subcommittee on medical care met under the auspices of the International Labor Organization in Montreal in 1943. On that occasion there was general discussion of a proposal "that all medical care services shall be regulated by law."

A delegate from Canada requested a definition of the word "regulated." He wanted to know if this term might extend to the actual rendition of medical treatment by physicians employed in the government service. Miss Laura Bodner, a member of the International Labor Organization staff, replied "it would." She elaborated by saying, "private voluntary insurance would be quite out."

In this discussion, the United States delegate, Mr. Isadore Falk, who took an active part, proposed an amendment to eliminate the word "regulated" as applied to medical care and substitute this definition of the topic; "the nature and extent of the care provided by the medical service shall be defined by law."

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*Presidential address to the Ouachita Parish Medical Society, December, 1946.

"The International Labor Organization medical program would relegate physicians to the level of mere functionaries. Under the Wage and Hour legislation, they would be classed simply as skilled workers. Private practice would be frowned upon. All medical service would be regulated by law. Administration would be by a central government agency with the help of unified local agencies. One recommendation calls for establishment of chairs of socialized medicine in the medical schools."

One provision of the International Labor Organization recommendations for worldwide socialization of medicine suggests that each national administrative headquarters of the medical services shall issue a series of pocket handbooks, "which show for the different medicines the most economical composition, dosage, form and method of dispensing."

Another section of the recommendations reads: "to prescribe unnecessary drugs or treatment does the patient no good and is a source of loss to the fund. Therefore, it should be made legally compulsory for the doctors to follow the rules for economical treatment in the interest both of the social insurance scheme and of the nation as a whole. The rules should give practical hints on treatment which will give the best results at the lowest cost."

Such is the path down which the people of the United States are heading and the degradation to which the medical profession is stooping if the recommendations of this group of ten or fifteen government payrollers in Washington, each drawing a salary of $4,000 to $12,000 a year from the Federal Government, are enacted into law. This group is headed by the following: Mr. Arthur T. Altmeyer, Mr. Isadore Falk and Mr. Wilber Cohen of our own Social Security Board. Presidential backing was secured by former Justice Samuel I. Rosenmann, formerly Special Adviser to the President.

While the Wagner-Murray-Dingell Bill, known as Senate Bill 1606, was dropped in committee at the last session of Congress, its proponents served notice that their fight had just begun. This places America at the crossroads and facing certain clear cut realities. On the one hand is the American concept of the inherent rights and sanctity of the individual, the human being. The Government exists merely to serve the individual who authorizes and supports it. The result is our freedom of enterprise society. On the other hand is the materialistic doctrine of the totalitarians. Their basic tenet is the ultimate supremacy of the State, with the individual existing solely to serve. They deny the existence of God and deprive man of dignity and separate status.

During the last twelve years we have witnessed the encroachments that are leading, even now, to a creeping paralysis of our social and economic functions. The designs of the schemers are made crystal clear. It is not merely a matter of medical care—or the regimentation of the medical profession that is at stake. It is our country, our beliefs, our concepts, our very souls that are involved.

Proponents of political medicine in this country have almost exclusively based their case and center their propaganda on the rejection statistics of the Selective Service System. The statement that 4,217,000 young men were rejected by selective service boards, as physically unfit, has been their spearhead in trying to show the American people that under our present system of medical care there has been a serious breakdown of the health of our nation. They failed to tell the public that these statistics are chiefly from the peace-time years when the Army was literally seeking out the very flower of America's young manhood—men who in every sense were mentally and physically perfect. To illustrate how particular we were during those days, I remember making a special trip to the Camp Livingston induction station to inquire about the extraordinarily high rejection rate for psychiatric reasons. Some of the questions asked the prospective soldiers were—"were you ever startled by an unexpected noise near you such as the back firing of an automobile"? If the answer was yes, the man was rejected as being mentally unfit. On testing for illiteracy they would ask such questions as "what is electricity"? "When was the first railroad built"? If they could not supply a satisfactory answer, the man was considered to be illiterate and thus rejected.

According to recent figures released by the War Department covering the period from January 1, 1942 to June 30, 1945, 1,750,000 men and women were rejected because of neuropsychiatric disorders, or mental illness, and the proponents of socialized medicine failed to state that the care of the mentally ill has been largely under the direction of the Government for many years. Obviously, the results are not very satisfactory. By what obscure reasoning can medicine be blamed for the result of bad environment, poor sanitation, housing, education and the lack of ordinary individual and community common sense. Mental deficiency and illiteracy make up another large group of rejections of which negroes, although called for induction in small numbers, contributed more than 44 per cent and this country has had compulsory education for many years. In their arguments these were also charged to inadequate medical care. Among those who were rejected for mental disease were some who simply lacked intelligence to become soldiers or sailors, and, in many instances, useful citizens. Among this group were the idiots, imbeciles, and the low grade morons.

Two hundred and eighty thousand men were rejected for reason of syphilis, and of this number 60 per cent of the rejectees were negroes. Treatment is available under Government sponsored and administered facilities for all of those who are unable to obtain private care—so it is hard to see how private medicine can be blamed for this condition. Three hundred and twenty thousand were rejected for musculo-skeletal defects such as a congenitally short leg, a club foot, a withered arm, or a dislocated hip. More than 445,000 were "manifestly disqualified." These included the armless, the legless, the totally blind, the mutes and others. It is obvious no kind, or amount, of medical care could have been of any value to this group.

In the course of their devious arguments for socialized medicine the proponents switched with great facility from the dark picture they had conjured of the nation's health as illustrated by the selective service data to paens of praise for the high standards of medical and surgical care afforded the men in military service. This argument can be completely deflated in two sentences; first, the same civilian physicians, who were so viciously maligned on one count, provided that admittedly high standard of military medicine, because the working medical officers in the service were almost exclusively civilian doctors who volunteered to serve their country during the emergency. Secondly, with the finest of equipment and supplies, one-third of all the physicians in the country gave excellent medical care to the healthiest and most fit, one-twelfth of our population. This brings to mind an incident which happened while I was on duty at Letterman General Hospital, San Francisco, California. The wife of a regular Army Major was brought in seriously ill, needing an emergency operation. When we advised him of this fact he immediately said he did not want any Army doctors operating on his wife. Inasmuch as we were under orders to be as nice as possible to everyone, I swallowed my first impulse to tell him to go take a jump in the lake and advised him that I was not any Army doctor—that I was one of the same civilians, whom he could take his wife to for the operation. He, like so many others, had not stopped to consider that we, who were doing the work, were not regular Army doctors.

According to one witness before the subcommittee last summer, and according to all available world information, the United

States has the finest hospitals, the best medical equipment and the most highly developed medical and surgical technics in the world. The complete revolution of medical practice, medical education, hospitals and research under Federally administered compulsory health insurance programs would bring about progressive deterioration and finally the collapse and destruction of the great gains which American medicine has made under our free medical enterprise system. This has happened whenever the system has been placed in effect.

The time has now come when every American—every believer in the American way of life—should join in unqualified opposition to this attempt to foist on this country this foreign ideology—this strictly collectivist mechanism — compulsory health insurance. We should be constanly on our guard and raise our voices against the tendency of foreign dictated bureaucracy to gain the powers of Government and make the people the servants thereof in order that we may return to the America for which we have fought and won two great wars at the cost of the lives of so many of our finest young men. Well may we recall the stirring admonition of Rousseau to his fellow countrymen of the eighteenth century, in France. "Free men remember this: Liberty may be acquired but once lost, it can never be regained." And well, too, may we recall the passage from the Gospel of St. Matthew—"Beware of false prophets, who come to you in clothing of sheep, but inwardly are ravenous wolves. By their fruits ye shall know them."

LIGATION OF THE VENA CAVA*

A CRITICAL EVALUATION BASED ON A STUDY OF 22 CASES

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In May 1942 we presented before this Society a preliminary report on the treatment of pelvic thrombophlebitis by ligation of the inferior vena cava and ovarian veins[1]. At that time three cases in which this procedure had been used were described in detail. Since then there have been 19 additional cases in which this operation was performed by members of the Department of Gynecology, Tulane University School of Medicine. Of this total of 22 operations (table 1), 18 were performed at Charity Hospital and four at Touro Infirmary.

TABLE 1

Indications

Pathology	Cases
Phlebothrombosis, common iliac vein associated with uterine fibroids	2
Suppurative pelvic thrombophlebitis (postoperative)	1
Suppurative pelvic thrombophlebitis (puerperal)	19
Total	22

As previously stated,[1, 2, 3] we believe that patients with postpartal or postabortal sepsis who fail to respond in a reasonable period of time to non-surgical measures should be seriously considered as candidates for ligation of the normal venous return from the uterus, that is, the vena cava and both ovarian veins. Prior to 1944 all patients were treated by means of a conservative regimen including chemotherapy[4, 5] and as soon as penicillin became available, it was used. When these measures failed, recourse had to be taken to operative intervention. Immediately after operation all chemotherapy and antibiotics were discontinued in order to determine the effectiveness of the operation itself and also to be sure that no other factor was responsible for the rapid drop in temperature and subsequent improvement. For an accurate evaluation of the efficacy of penicillin and sulfonamides, in addition to other therapeutic measures used in the non-surgical management of these cases one must take into consideration a number of factors. The interval between the onset of intra-uterine infection and the administration of sulfonamides and penicillin has a direct bearing on whether or not the patients will be benefitted and there is also the possibility of the infecting organism being "sulfa fast" or penicillin resistant.

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*Presented before the regular monthly meeting of the Orleans Parish Medical Society, December 9, 1946.

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Though the incidence of puerperal sepsis has been lowered in the past ten years, this disease still ranks high as a cause of maternal death and despite the use of penicillin and sulfonamides we still see cases of suppurative pelvic thrombophlebitis in which surgical therapy is not only indicated but urgent. At present, on our service, all patients with postpartal or postabortal sepsis are treated by a conservative plan including large doses of penicillin and sulfonamides. However, if the patient fails to improve at the end of 72 to 96 hours following the institution of therapy or if a pulmonary infarct develops during this therapeutic regimen, the vena cava and ovarian veins are immediately ligated. If the patient enters our service with a postpartal or postabortal infection and pulmonary infarction is already present, she is operated on immediately without any preliminary treatment with sulfonamides or penicillin.

In addition to suppurative pelvic thrombophlebitis, we believe that ligation of the vena cava is indicated in cases of phlebothrombosis if the clotting process has extended into the external iliac or common iliac veins. If we are to lower the incidence of pulmonary embolism in gynecologic and obstetric cases, we should take cognizance of the fact that unilateral edema in a pregnant patient or one with a large ovarian or uterine tumor should not be ascribed to pressure of the growth until the possibility of phlebothrombosis or thrombophlebitis has been eliminated. Investigation of these cases preoperatively by venography and inspection of the pelvic veins at operation have convinced us that edema of the leg can be and most probably is due to a phlebothrombotic process in one of the large tributaries of the vena cava. A review of the deaths from pulmonary embolism occurring at Charity Hospital during the period from July 1, 1939 to January 1, 1946, revealed that in 50 autopsies from the gynecologic and obstetric services, fatal pulmonary emboli and phlebothrombosis were found in 12 cases.[3] Six of these occurred following operation on the female pelvis, particularly hysterectomy for uterine fibroids. More important than the deaths from pulmonary emboli following operation is the startling fact that five of the fatal emboli occurred in patients who were not operated on but who were being prepared for operation for a pelvic tumor associated with edema of the lower extremity or extremities, the latter probably assumed to be due to pressure by the growth. In the light of our present day knowledge of phlebothrombosis these deaths might have been prevented. In contrast to these five cases of pulmonary emboli there were three patients admitted to the Tulane Gyne-

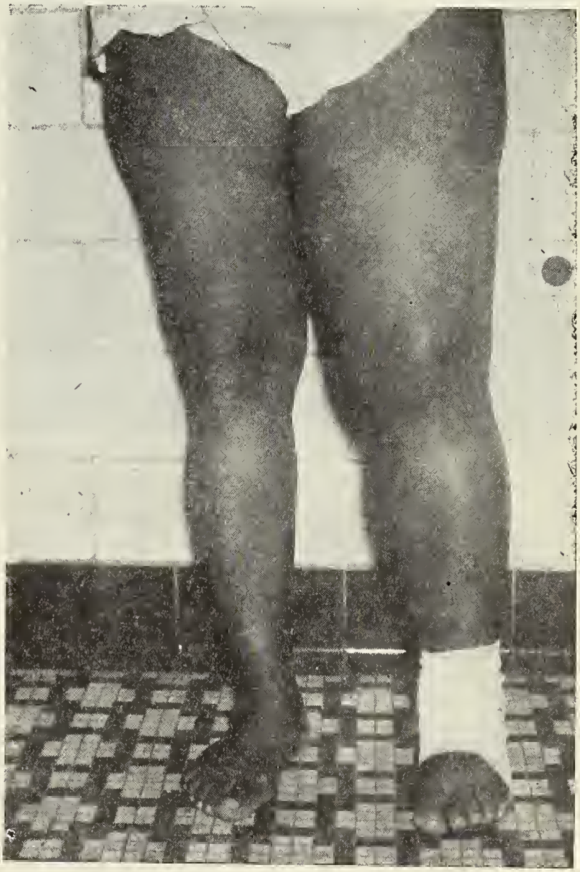

Fig. 1. Patient with unilateral edema and large uterine fibroid. Phlebothrombosis of veins of left leg extending to formation of inferior vena cava.

cologic Service at Charity Hospital in New Orleans during the same period with the same syndrome, in whom phlebograms confirmed the presence of phlebothrombosis and in whom recovery followed venous ligation prior to removal of the uterus. Recovery without ill effects followed ligation of the vena cava and hysterectomy in two patients and ligation of the superficial femoral vein and hysterectomy in the third patient. We should be very careful then to ascribe unilateral or bilateral edema to pressure from an intra-abdominal growth. We believe that many of these cases can be explained on the basis of a phlebothrombotic process. In these cases, ligation of the involved veins above the thrombus is the first step to the successful surgical attack on the abdominal tumor.

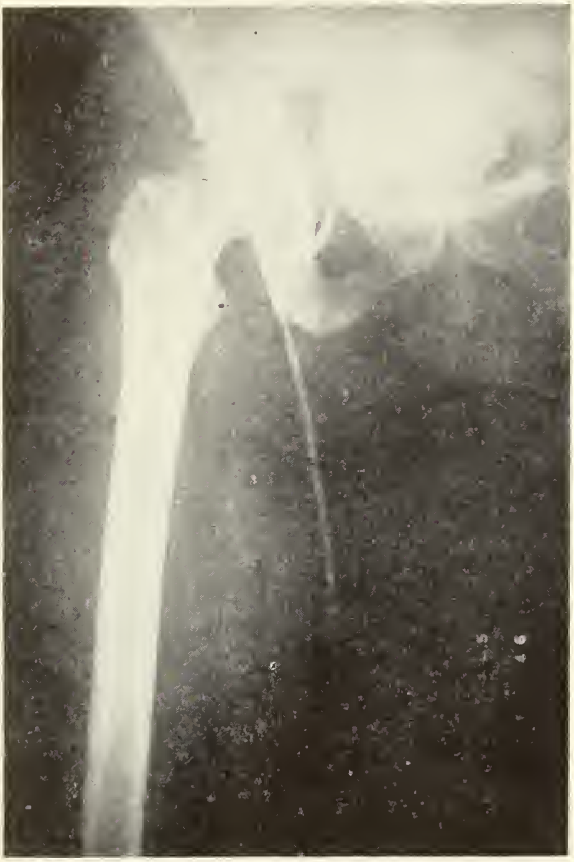

Fig. 2. Phlebogram of same patient, showing absence of deep venous return. Note extensive venous collaterals in region of pubic bones.

Though many reports on the ligation of the vena cava have appeared in the literature in the past two years and though some authors advocate the extroperitoneal approach, it must be realized that ligation of the vena cava in the cases reported by them was performed on veins of the lower extremities or pelvis in men as well as women.

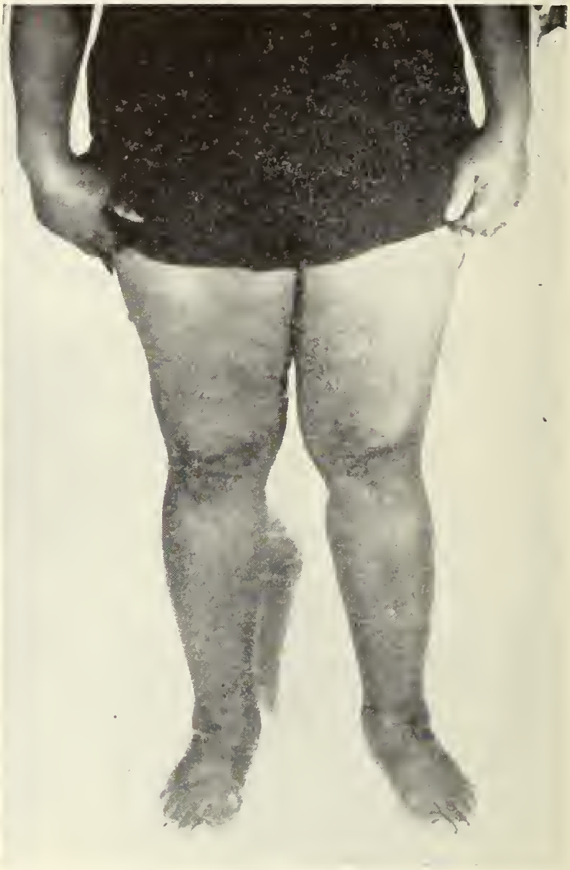

Fig. 3. Same patient three weeks after ligation of inferior vena cava and hysterectomy. Ankle edema still present but this has disappeared at six month follow-up examination.

In the treatment of suppurative pelvic thrombophlebitis in women, it is important not only that the vena cava be ligated but also the ovarian veins as the disease most frequently involves many pelvic veins,[6] that is, the uterine, hypogastric or iliac veins, in addition to some involvement of the ovarian veins. Therefore, the successful surgical management of suppurative thrombophlebitis of the pelvic veins requires ligation of the normal venous return from the uterus, that is, the vena cava and ovarian veins. Ligation of the ovarian veins alone or one ovarian vein and one iliac or the other, does not produce the high percentage of successful results that ligation of the normal return of the venous flow from the uterus does.[3, 7, 8, 9] This can best be accomplished by a transabdominal approach since it offers easy access to the whole of the pelvis; the pelvic veins can be thoroughly explored, the ovarian veins ligated above any clotting process found, and the vena cava ligated at the same time.

Patients with suppurative pelvic thrombophlebitis are acutely ill at the time of operation; they usually have temperatures of from 103° to 106°F. daily and chills, and in all of our cases there was infarction of the lung. Despite the rapid pulse rate and high temperature, these patients tolerate the operation extremely well. Spinal anesthesia was used in all our cases except one in which local anesthesia was employed.

TECHNIC

A para-umbilical midline incision is made, the recti are separated in the midline, and the peritoneal cavity is opened. The uterus, ovaries and tubes as well as the ovarian, uterine, hypogastric and iliac vessels are palpated. The peritoneal reflection of the lateral colic gutter is incised and the cecum and ascending colon are then reflected medially. These structures are mobilized to the midline. The right ureter and right ovarian artery and veins are identified. The right ovarian veins and artery are ligated above the clotting process. If a clotting process is not found on the right side, the veins and artery are ligated anyhow. The vena cava is then identified and dissected out by means of sharp and blunt dissection. Two ligatures of cotton crochet No. 20 are applied around the vena cava about an inch apart and about the level of the fourth lumbar vertebra. These are tied tightly. The right lumbar sympathetic chain is then identified and severed. The cecum and ascending colon are then replaced in normal position and peritonealization is accomplished. The descending colon and rectosigmoid are then mobilized by means of an incision along the peritoneal reflection of the left colic gutter and are retracted medially. This exposes the left ovarian artery and vein and left ureter. The left ureter is identified and as on the right side, the left ovarian veins and artery are ligated above the clotting process. If no clotting process is found, they are ligated anyhow. The aorta is retracted medially and the left lumbar sympathetic chain severed. The sigmoid and descending colon are then replaced and peritonealization accomplished. Finally, the abdomen is closed in anatomic layers. If at the time of operation it is considered unwise to sever the sympathetic chains on the right and left sides either because of the patient's condition or because they are not easily located, then bilateral lumbar sympathetic block with procaine hydrochloride is performed daily after the operation for four or five days. The sympathetic nerves are sectioned or sympathetic block is done in order to eliminate as much as possible spasm of the vessels in the extremities which might be occasioned by ligation of the cava. We believe that this is an important factor in the management of the case and is one reason why we see very little or no edema in our cases postoperatively.

POSTOPERATIVE RESULTS

In judging the efficacy of the surgical management in our cases of suppurative pelvic thrombophlebitis one must remember that ligation was performed only in cases which failed to respond to all nonoperative measures advocated for this condition and that these patients were the most acutely ill of all cases of puerperal sepsis which we have encountered. In the two patients in whom ligation was done for phlebothrombosis of the common femoral vein associated with uterine fibroids and the one case of postoperative suppurative pelvic thrombophlebitis there were no deaths (table 2).

TABLE 2
Mortality
(Uncorrected)

Pathology	Cases	Deaths
Phlebothrombosis	2	0 (0%)
Suppurative thrombophlebitis (postoperative)	1	0 (0%)
Suppurative pelvic thrombophlebitis (puerperal)	19	4 (21%)
Total	22	4 (18%)

Note: All patients dying within one year after operation are included as operative deaths.

In the 19 patients with puerperal suppurative pelvic thrombophlebitis who were operated on, there were four operative deaths (table 2). This is an uncorrected figure, since all patients dying within one year of the time of operation were considered as postoperative deaths. The first death (table 3) occurred in 1942, two hours after ligation of the vena cava. The patient when first seen had peritonitis and was hopelessly ill but she was operated on immediately and in addition to peritonitis there was thrombosis of the left vena cava and terminating in a coil in the auricle of the heart. The second death (table 3) occurred in 1944. The patient was operated on some time after she had been in the hospital and died six days later, not immediately as a result of the operation but from septic infarcts with formation of abscesses in the liver, lungs, spleen and kidneys, and endocarditis of the heart. Obviously, this patient was operated on too late. The third and fourth deaths (table 3) occurred three and three and a half months respectively, following operation. The deaths in these two cases can be attributed to the fact that the patients were operated on later than our surgical judgment deemed it wise. Since we were trying to evaluate the efficacy of penicillin and sulfonamides in the therapy of postpartal and postabortal sepsis in these patients, operation was deferred much longer than is our usual practice. Though the operations were successfully performed, these patients died from secondary implants elsewhere in the body, one from the mycotic aneurysm of the inferior mesenteric artery which ruptured and the other from bacterial endocarditis. These two cases do prove, however, that penicillin and sulfonamides in huge doses do not cure all cases of postabortal or postpartal sepsis. All other patients survived the operation for at least two years and though all patients are not living today, these deaths were in no way the result of ligation of the vena cava or suppurative pelvic thrombophlebitis. One patient died two and a half years following the operation from hemorrhage during a thoracotomy being performed for persistent pulmonary abscess which was a result of the original infarction. A second patient died three years after operation from hemorrhagic encephalitis following arsenical therapy for syphilis. A third patient died three and a half years after operation from third degree burns.

TABLE 3

Deaths

Case	Time Following Operation	Cause
1	2 hours	Peritonitis at time of operation. Thrombus extended from uterus, ovarian vein (lt.). renal vein, superior cava with auricle of heart.
2	6 days	Septic infarct with abscess formation in heart, spleen, lung, kidneys.
3	3 months	Mycotic aneurysm of inferior mesentery artery.
4	3½ months	Endocarditis.

All patients living today have been examined periodically by us and by Drs. Burch and Winsor,[10] and Ray[11] of the Department of Medicine, Tulane University. Not only have the appearance of the extremities and function of the genital tract been studied but the circulation in the extremities has been thoroughly investigated. Dr. Burch and his co-workers concluded,

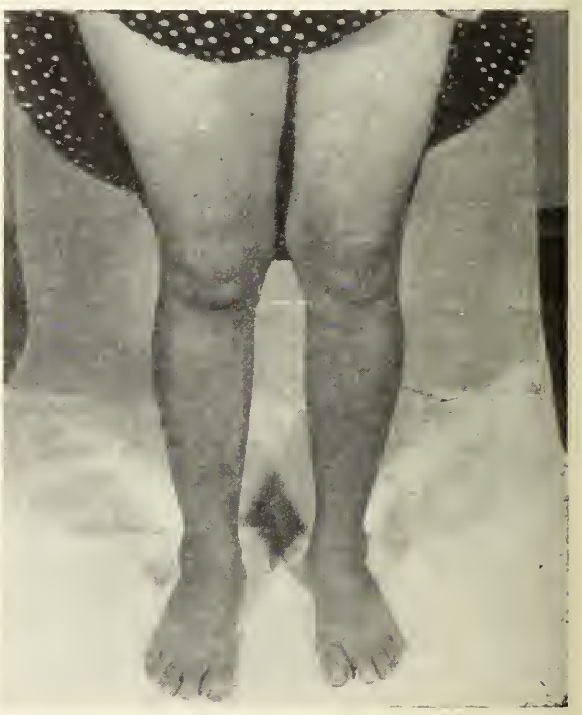

Fig. 4. Extremities of patient four years after ligation of inferior vena cava and ovarian veins for puerperal suppurative pelvic thrombophlebitis.

"All patients showed remarkably good compensation of the circulation following ligation." All these patients are able to perform any type of work, are not restricted in any capacity and feel well. Their occupations vary from those of housewife to farming. In one or two cases slight edema is occasionally present but this is negligible. One patient has had two babies since the operation and has had no complications either antepartally, intrapartally or postpartally. Two other patients in this series have become pregnant and miscarried spontaneously without any complications. There has been no change in the patients' menstrual cycle either in duration, frequency or amount. In one case bleeding persisted from retained secundines but this was corrected by curettage. One patient had a third degree burn of the leg necessitating skin grafting and the operative note and follow up on the patient showed that the skin graft took just as easily as if the vena cava had not been ligated previously.

CONCLUSION

Ligation of the vena cava carries a low operative mortality. The end results and the appearance and function of the patients' extremities are very good. The procedure is one that should be used without hesitation when indicated. In gynecology and obstetrics, we believe that it is indicated in cases of phlebothrombosis of the iliac vessels whether the clotting process occurs preoperatively or postoperatively. It is also indicated in cases of postabortal and postpartal puerperal sepsis if conservative therapy is ineffective, if infarction to the lung develops, or if the patient has an infarct to the lung from a suppurative pelvic thrombophlebitis when first seen. The minimal amount of edema and circulatory disturbance occasioned by vena cava ligation in our cases, we believe, is due to the fact that at the time of operation the sympathetic chains are sectioned or if this is not possible at operation routine blocking of the lumbar sympathetics is performed, bilaterally, daily, for five or six days after operation.

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DISCUSSION

Dr. L. S. Robinson (Winnsboro): A principal concern of surgeons has always been the establishment of a sufficient collateral circulation following ligation of any large venous trunk, especially the inferior vena cava. However, it has been proved in numerous cases that life is compatible with ligation or obliteration of the inferior vena cava. There is an abundance of experimental and clinical evidence which demonstrates the ability of the circulatory apparatus to adapt itself quickly to either sudden or gradual complete obstruction of the inferior vena cava. A prevalent older belief was that sudden ligation of the inferior vena cava is incompatible with life and that the inferior vena cava should be ligated in two stages to allow for gradual establishment of the collateral circulation. However, more recent observations prove that the vessel may be safely ligated at one stage at any level below the renal veins.

In 1909 Houzel stated that ligation of the inferior vena cava was indicated if the vessel were wounded or had to be resected to remove a tumor, and was without great danger. Pfaff, in 1926, concluded that ligation of the inferior vena cava below the renal veins was safer than attempting to suture the wounded vessel. Ligation of the inferior vena cava has gradually become increasingly popular since about 1906 in the surgical treatment of septic thrombophlebitis of the pelvic veins and deep veins of the legs.

There has been but little experimental work using the human cadaver in the study of the collateral circulation of the inferior vena cava. In an effort to study the collateral circulation, during the autopsy of a case with obliteration of the inferior vena cava from the renal veins to the common iliac vessels, Sappey, in 1862, injected the venous system with tallow via the femoral and axillary veins. He divided the collateral circulation into anterior, lateral and posterior channels, and presented an excellent description of the veins concerned and of their communications. Batson's work (1940, 1942) on the function of the vertebral veins and their role in metastasis emphasized the potentialities and the collateral communications of this system in the cephalad flow of blood. He has performed a very complete study of the vertebral veins in 100 cadavers by injecting a radiopaque material into the dorsal vein of the penis. More recently Northway and Greenway (1944) studied the collateral circulation following ligation of the inferior vena cava at a point on the lower one-third of the vessel. In their experiments they used unprepared cadavers and injected the venous system via the internal saphenous vein with a thin suspension of red lead. Their results further demonstrate the importance of the vertebral plexus of veins as collaterals following ligation of the inferior vena cava.

Considerable time and effort has been spent throughout the years in experimental work on animals in an effort to study the collateral circulation following ligation of the inferior vena cava at different levels. It is generally conceded that the inferior vena cava and its connections are essentially similar in dog and in man, and most of the experimental work has been done on this animal.

In 1669, Lower published the first account of experimental ligation of the inferior vena cava in an animal.

Brunton (1883) cited Ranvier's experiments of ligating the vena cava of the dog and the observation that it did not always produce edema of the legs.

Purpura, after a great number of experiments on the dog concluded in part that life is possible after ligation of the inferior vena cava at a point between the common iliac and hepatic veins, but more difficult to maintain if the ligation is made above the renal veins.

Gosset and Lecene, in 1904, and Leotta, in 1907-8, stated that the inferior caval system in dog is absolutely comparable to that of man; that ligation of the trunk of the inferior vena cava below the renal veins is not accompanied by any pathological phenomenon, not even a trace of edema of the legs; and that ligation of the trunk of the inferior vena cava above the renal veins provokes grave lesion of the kidneys.

Offergelt, in 1907, reported experiments on ligation of the inferior vena cava in its lower one-third on rabbits, cats and dogs. All of the cats died, but the other animals survived without edema or severe disturbance in their sensitivity or temperature. He noted only slight extension of cardiac dullness to the left and a transitory tremor of the legs.

Bejan and Cohn, in 1911, did similar experiments on the dog. They concluded that ligations of the inferior vena cava below the renal veins, or at the attachment of the renal veins and including one of the veins in the ligature, or immediately above the renal veins and the left suprarenal vein are compatible with good health. However, their ligations at levels higher than the right suprarenal vein always resulted in death.

In 1929 Polkey studied the phenolsulphonephthalein output after ligation of the vena cava below the renal veins, in dogs, and found the kidneys functioning normally.

Experimental and clinical investigations on ligations below the renal veins have shown that edema or slight albuminuria may develop, but neither is common, and both are usually transitory. Most authors agree that the mortality is high and the kidneys are seriously affected after ligation of the inferior vena cava above the renal veins. However, a minority may recover and the kidneys resume good functional activity.

Batson's injection experiment on the monkey has done much toward demonstrating part of the potential collateral circulatory system and the importance of the vertebral veins following ligation of the inferior vena cava.

As may well be expected, the veins concerned in the collateral circulation following ligation of the inferior vena cava are numerous. Haller, in 1756, was probably the first to describe the collateral channels in a case where the lower third of the inferior vena cava was obliterated. In his case the circulation was carried on by the superficial abdominal veins, the right ureteral vein, and the right ovarian vein.

Baillie, in 1793, described the collateral circulation by the lumbar and azygos veins, but the thrombosis involved the upper two-thirds of the inferior vena cava.

Cruveilhier showed that one of the principal means of establishing circulation between the inferior vena cava and the superior vena cava are the azygos and vertebral system of veins. He mentioned that the great azygos vein, which is generally regarded as the chief means of communication between the two vena cavas, is not indispensable, because of the presence of the vertebral plexus of veins.

Wilson, in 1804, described the vertebral. portal and azygos routes in detail.

Bidault, in 1804, and Castelnau, in 1841, described the superficial veins making up the collateral circulation in cases of obliteration of the lower third of the inferior vena cava. Hodgson also described the superficial veins in a similar case, exhibiting in addition dilation of the lumbar veins, the internal mammary veins and the deep epigastric veins.

Hallet described a case of obliteration of the inferior vena cava in a woman, and stated that the veins of the uterus were dilated and formed a plexus to aid it, of which the veins of one side communicated with veins of the opposite side. He found the collateral circulation to be reestablished by the uterine veins, the left utero-ovarian vein, the left ureteral vein and the left renal vein.

Sappey and Dumontpallier, in 1862, reviewed the literature and described the collateral circulation in different types of obstruction of the inferior vena cava. Their description of the collateral circulation following obliteration of the lower third of the inferior vena cava included the superficial and deep veins of the anterior abdominal wall, intercostal veins, veins of the lateral abdominal wall and thorax draining into the axillary veins, and the "posterior currents" which included the lumbar veins and the azygos system, the portal system via the inferior mesenteric vein, and the vertebral system of veins. Nicaise quoted Sappey as finding that if a ligation was made at a point on the trunk of the inferior vena cava and the external iliac vein was injected, that the fluid easily arrived in the heart, and that the amplitude of the anastomotic paths which supply this venous trunk was great enough that one could annihilate the trunk and the injected liquid would reach the heart. The venous anastomosis would also connect the two ends of the inferior vena cava.

Thomas, in 1893, showed in table form the veins involved in the establishment of the collateral circulation following various types of obstruction of the inferior vena cava.

Poirier stated that the collateral circulation following ligation of the inferior vena cava was extensive, and included the azygos veins, lumbar and sacral veins, diaphragmatic veins, vertebral veins, renal veins, veins of the anterior abdominal wall, and finally the portal vein by its numerous anastomoses.

According to Simpsin (1924) the absence of signs of serious vascular upset in cases of complete and sudden obstruction is probably due to the multiplicity of small collateral channels, all of which are capable of sudden slight dilation, the sum total of which is sufficient to establish rapid compensation. The period of adjustment is indicated by the transient edema.

Batson's (1940, 1942) experiments on the monkey and on the human cadaver demonstrated a set of valveless, plexiform, longitudinal, venous channels that join the pelvic veins to the cranial venous sinuses without the intermediation of the lungs. The verebral vein system parallels, connects with, and provides by-passes for the portal, the pulmonary, and the caval systems of veins.

In 1944 Northway and Greenway found the vertebral plexuses to form an adequate communication between different parts of the inferior caval system as well as between the inferior and superior caval systems. They concluded that this extensive system is more than adequate in its collateral potentialities, so that ligation of the inferior vena cava merely serves to place a valve within the vena cava, with that portion of the vein proximal to the ligature being readily filled from the communicating branches of the vertebral plexus.

Recently, in the Department of Anatomy at Tulane, the collateral circulation was studied following ligation of the inferior vena cava at a point on the lower third of the vessel. Stillborn infants were injected with radiopaque Neoprene, a synthetic liquid latex, and the collateral circulation was studied by x-ray, dissection, and corrosion methods. The use of radiopaque Neoprene, for injection studies was satisfactory for dissection and corrosion, but failed to give a good outline of the veins in the roentgenograms. The use of corrosion specimens for the study of the collateral circulation showed an abundance of collaterals, but identification of many of the veins gave the best results, by far, of any method employed in the study.

Observations during the injection of Neoprene into the saphenous or femoral veins showed the vertebral and azygos systems to fill immediately. Too, the visceral veins of the abdomen and pelvis filled readily as evidenced by the injection of the mesenteric veins, the liver, and pelvic organs. Studies following injection showed the vertebral plexus of veins, the veins of the neck, the veins of the thoracic wall (including the azygos system), the veins of the wall of the abdomen and pelvis, and the veins of the abdominal and thoracic viscera to be well filled. However, the superficial collaterals were found to be only partially filled, if at all, and anastomotic connections, as evidenced by the injection mass, were frequent.

These results coincide with clinical and experimental evidence concerning surgical ligation of the inferior vena cava, that is, (1) the vertebral, azygos, and portal systems of veins play a large role in the collateral circulation following ligation of the inferior vena cava, the superficial veins being rarely dilated; (2) following ligation of the inferior vena cava at any point in the lower third the proximal and distal ends still communicate by way of its vast collateral system; (3) after ligation of the inferior third of the inferior vena cava the collateral channels are adequate for return of blood from the lower portions of the body to the heart.

Dr. B. Bernard Weinstein (New Orleans): The past few decades has brought many advances in asepsis, antisepsis, chemotherapy, and in atraumatic obstetrical and gynecological surgery, yet pelvic thrombophlebitis is an all too frequent puerperal, postabortal and postoperative complication. It is characterized by a high mortality and has shown poor response to chemotherapy and conservative measures.

Hunter, in 1784, was the first to apply the principle of proximal venous ligation to control suppurative thrombophlebitis. Freund nearly a century later (1898) was apparently one of the first surgeons to apply the method to gynecology. In two cases he ligated and excised thrombosed ovarian veins, and the broad ligament. Both patients died. In the early portion of this century the work of Braum, Trendelenberg and others popularized the method which reached some degree of popularity about 1912 when C. Jeff Miller, reporting before the American Medical Association, was able to report seven cases of successful ligation of the iliacs or vena cava in pelvic thrombophlebitis. Subsequently Dr. Miller had several failures and became discouraged and abandoned the method. About this time after a slight flurry of popularity which extended from 1900 to 1915 the method passed out of use. The reason for the failures in Dr. Miller's cases was that the venous return from the uterus was only partially ligated. If one appreciates the necessity of bilaterally ligating the pampiniform plexus or its equivalent the ovarian vein, and the uterine drainage proper that is uterine veins, hypogastric veins, common iliac veins or inferior vena cava depending on the extent of the thrombosis, one can readily see why inadequate ligation would lead to the possibility of further propagation of thrombi or dissemination of emboli leading to death. It is far more astounding that the early inadequately ligated cases lived than that the method failed.

By 1937 Krosotski was able to report 40 successful instances of ligation of the inferior vena cava. If one will bear in mind the adequate collateral circulation which is established and which Dr. Robinson so beautifully demonstrated, and the fact that one must ligate inferior to the renal veins, the ligation of the inferior vena cava *per se* is not a formidable procedure.

In 1940 Dr. Conrad Collins faced the problem of high mortality in cases of septic pelvic thrombophlebitis, reviewed the situation carefully and critically, and decided to attempt the total ligation of the venous return from the pelvis in these cases, that is a ligation of the inferior vena cava somewhere above the area of thrombosis and below the renal veins, and bilateral ligation of the ovarian veins. The success of Dr. Collins and his associates in their first 12 cases which I was privileged to follow very closely stimulated my own interest in the problem, and lead to my successful use of the procedure in four cases.

This procedure, which is ostensibly radical, is actually conservative and lifesaving. As Dr. Collins has told you, the procedure has now been performed more than 20 times by various members of the Gynecological, Obstetrical and Surgical Staff of Tulane University (including the cases of Dr. Collins) with one death. This one death occurred in a woman who was moribund at the time of operation, and who should have been left out the series, but it was the surgeon's opinion it was far better to try a final attempt to save the woman's life than to keep his mortality statistics pretty.

The diagnosis of pelvic thrombophlebitis is an extremely difficult one, and must frequently be made by exclusion of kidney, bladder, wound and lung infections. Very frequently the diagnosis is missed until the first shower of emboli occurs. Then the diagnosis is frequently missed unless one has available a roentgenologist who appreciates the pulmonary picture of multiple infarction and differentiates it from bronchopneumonia with which unfortunately it is not infrequently confused. However, even after the occurrence of the first shower of emboli ligation if immediately performed, will be successful as indicated by three cases in Dr. Collins' series.

The use of postoperative lumbar sympathetic block or of section or excision of the lumbar sympathetic chain at the time of operation is extremely beneficial in promoting the rate of development of collateral circulation. After venous ligation of the pelvic veins the patient who has not had an excision of the lumbar sympathetic chain or a section of it, should have bilateral block daily for several days.

One of the surprising facts associated with the follow-up in these cases is their failure to develop edema of the lower extremities, or their development of an extremely mild and transient edema. This is attributable to the rich collaterals which had begun to develop as a result of the thrombosis present and which was subsequently further developed by the excision of the lumbar sympathetic chain or the use of lumbar sympathetic blocks following the ligation of the pelvic veins.

Collins is to be congratulated on both the courage he has displayed in tackling this difficult problem, and on the splendid results he has achieved. It points the way to a markedly greater salvage in this once fairly hopeless group of cases. Dr. Robinson has done a painstaking and beautiful piece of research which has many clinical applications. I am grateful to him for presenting his work before our Society, and I appreciated the privilege of hearing his paper.

THE UNSTABLE COLON*

ITS ROLE AS A PRECURSOR TO DIVERTICULOSIS

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In 1830, John Howship wrote a small

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*Read before the Ascension Parish Medical Society, October 31, 1946.

book entitled, "Practical Remarks on the Discrimination and Successful Treatment of Spasmodic Stricture in the Colon Considered as an Occasional Cause of Habitual Confinement of the Bowel." The ailment, according to Howship, was due to "a deficient freedom of relaxation in some part of the intestinal canal." Since then, much has been written regarding this subject, and unfortunately, because of its uncertain etiology, pathologic physiology, and confused classification, there exists in many instances no clear-cut concept of its true character, as may be recognized at present.

The terms, spastic, irritable (Sippy, Jordan, Kiefer), or unstable (Kantor) colon would seem to be justified in view of the neuromuscular nature of the disease and in the absence of actual inflammation of the colon. At this point, I am in sympathy with the classification of Bockus and Willard who have seen fit to classify the functional disorders of the colon, and follows:

COLONIC NEUROSES (NEUROMUSCLAR AND NEUROSECRETORY COLONOPATHIES)

I Motor neurosis: disturbances of tonus and propulsive motility.

(a) Spastic colon, chronic colonospasm, irritable or unstable colon.

(b) Nervous or emotional diarrhea.

(c) Congenital or neurogenic megacolon.

II Secretory neurosis: neurogenic mucous colitis, mucomembranous colic.

III Mixed neurosis: neurogenic disturbance of both the motor and secretory functions of the colon.

Classification has two major groups, colonic neurosis and simple constipation.

SIMPLE CONSTIPATION

It would appear, therefore, that while an individual may exhibit definite evidence of a mixed neurosis, that nevertheless in many instances examples of either the motor or sensory neurosis may be seen. Thus, it is possible to distinguish within reasonable limits the purely motor disturbance, that is, the unstable colon, from the secretory type, neurogenic mucous "colitis", and further, to recognize the co-existence of both in other cases. The term "mucous colitis", of course, is undesirable because of the absence of inflammatory process in this condition.

According to Bockus, a colonic neurosis may be defined "as an intermittent or constant disturbance of colonic function which cannot be ascribed to an organic or nonneurogenic constitutional disorder and which seems dependent upon a derangement of the autonomic innervation of the colon." The specific etiologic factor or factors, as well as contributory influences, of the colonic neuroses are not definitely known, nor whether similar or different factors serve to produce the various neuroses. However, the unstable colon may be considered as a constitutional condition, frequently characterized by symptoms and signs of a labile vegetative nervous system, and more specifically as a neuromuscular or motor disturbance of the entire alimentary tract, especially prominent in the colon, and emanating through the medium of a deranged autonomic nervous system, perhaps directly influenced from the diencephalon, or indirectly stimulated by one or more factors, such as psychogenic, endo-

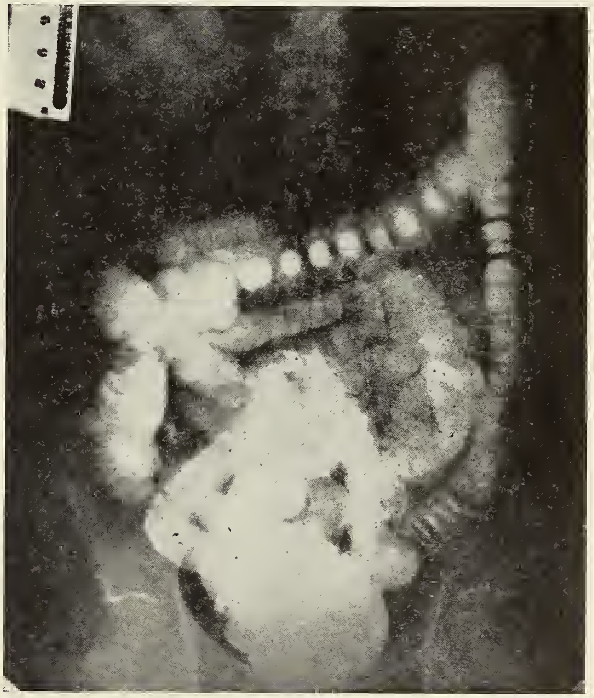
Fig. 1. Irritable colon (when first seen)

crinal, allergic, bacterial, and faulty intestinal hygiene.

The unstable colon is of necessity recognized, first by a process of elimination, secondly by the presence of frequently associated symptoms and signs, and finally and more conclusively, by the rather commonly occurring roentgen findings. While evidences of spasticity may be observed in any part of the alimentary tract, one particularly finds these areas in the colon, and especially in the descending colon, and further, the site of spasticity may be observed to change from time to time. The barium meal may be observed to pass through the colon very rapidly and though the shadow of the filled colon varies markedly, one of the most common findings is that of an abnormal narrowing of some part of the colon, especially the descending and pelvic portions; in addition, this area often presents little or no haustral markings; in some cases, the narrowed, spastic colon with slight residual haustrations reveals the "string of pearls" appearance. In this particular case, we are privileged to observe "the fine, shallow, irregularly arranged saw-tooth type of colonic margin described by Spriggs and Marxer as prediverticulosis," and yet, when present, said to be characteristic of the unstable colon. And now, it is fascinating to observe in this same patient eight months later, the development of diverticula at the apices of the serrated colonic border. Of interest, also, is the presence of the "string sign" of mucous colitis. Thus, we have observed, at first, an individual showing essentially roentgenographic evidence of unstable colon, and finally, the same individual at a later date, presenting definite roentgen indications of a mixed colonic neurosis plus diverticulosis. Still later, this individual may, of course, add to these functional and pathological abnormalities in kind, and by incurring the well-known complications and sequelae of diverticula.

Finally, it is of interest to note that this kind of diverticulum is known as the acquired, or false type. True diverticula, or the congenital type, contain all the coats of the intestine, whereas false diverticula are pouches of mucosa and serosa. Acquired or false diverticula usually occur at middle age or later, and are especially prevalent in the descending and pelvic colon. Therefore, it is conceivable that numerous factors associated with the ageing individual, such as constipation, incorrect bowel hygiene, repeated intestinal trauma, the hyperstimulation of the nervous system by modern civilization, and the unstable colon, may serve as factors in the development of diverticula in the presence of some weakness in the bowel wall, as a result of the increased intracolonic pressure common to these conditions. Under such circumstances as mentioned, the elastic mucosa may be allowed to herniate through the muscular fibers of the colonic wall and bulge outward before it the equally elastic serosa producing a globular appendage with a constriction or neck as it emerges through the muscular layer, thus producing the typical acquired, or so-called false, diverticulum.

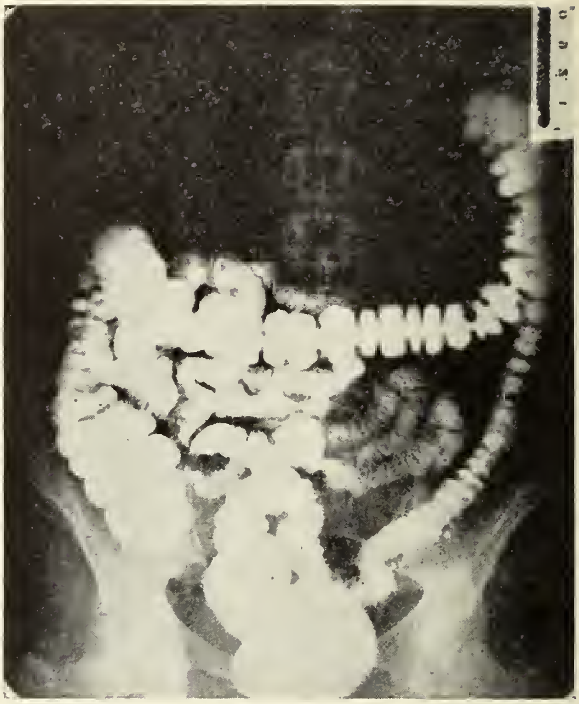

Fig. 2. Irritable colon, several months later, showing development of diverticula.

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ACTINOMYCOSIS

A REPORT OF TWELVE CASES WITH SPECIAL REFERENCE TO A MEDIASTINAL CASE

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The purpose of this paper is to present a brief summary of 12 cases of actinomycosis observed at the Charity Hospital at Shreveport, Louisiana, since 1938. One case in which the posterior superior mediastinum was involved and diagnosed by aspiration biopsy is presented in brief detail.

HISTORY

Harris and Priestley[3] state that Bollinger in 1877 first described the disease of cattle known as lumpy jaw. He attributed the cause to the organism *Actinomyces bovis.* In 1878 Israel reported a case in man. The following year Ponfick showed the essential similarity of the organisms causing these conditions in man and cattle. Lord in 1910 demonstrated that Actinomyces pathogenic for guinea pigs could be found in the gums and tonsils of apparently normal individuals.[4]

Fifty-eight cases of actinomycosis in America were collected by Ruhrah in 1899.[5] Erving brought this number up to 100 in 1902 and Sanford collected almost 700 unreported cases in 1922.[5]

DEFINITION AND ETIOLOGY

Lynch and Holt,[1] quoting Topley and Wilson, state that the term actinomycosis now applies to two groups of diseases of man and animal in which Actinomyces-like organisms are encountered. In the first group the proliferation of an anaerobic organism such as *Actinomyces bovis* gives the appearance of colonies which have a ray structure in pus or tissues and often are described as sulfur granules or "Drusen." In the second group are included all aerobic filamentous branching organisms that form a felted mycelium without any radial arrangements and without granule formation. Two type species are; (1) *Actinomyces graminis* which is not acid-fast and forms no granules in tissues and (2) *Actinomyces asteroides* (*Nocardia asteroides*) which is acid-fast and forms no granules in tissues. It is often the cause of pulmonary and brain lesions. According to Knott[7] the organism *Actinomyces asteroides* is capable of producing a pulmonary pseudotuberculosis in man and laboratory animals.

Walker and Hamilton,[2] quoting Zinzer and Bayne-Jones, define actinomycosis as an infectious graulomatous disease characterized by the destruction of tissues, suppuration, and overgrowth of fibrous tissue.

Harris and Priestley[3] state that actinomycosis really connotes a group of diseases just as the Actinomyces really belong to a group of organisms rather than a single pure strain. Some are aerobic and others are anaerobic. Most of them take the Gram stain and a few are acid-fast. Those that are anaerobic are referred to as Actinomyces and those that are aerobic with a tendency to reproduce by fragmentation of the mycelium are referred to as Nocardia.

Cecil[4] states that the organism occurs in pus or tissues as lobulated or spherical granules, which vary in size from very minute particles to forms about 1 mm. in diameter. *Actinomyces bovis* is anaerobic and grows best at 37° C. in glucose agar in a zone 5 to 10 mm. below the surface and *Actinomyces asteroides* is aerobic and grows on ordinary laboratory media at room and incubator temperatures.

PATHOGENESIS

Two views exist as to the mode of infection. One is that the organisms gain entrance by way of grasses and grains that are ingested and the other that the organisms exist as saprophytes in the intestinal tract, particularly the mouth, and gain entrance under the proper conditions of lowered resistance and a portal of infection, then becoming pathogenic. Actual facts do not substantiate the general belief that the disease is contracted by contact with cattle afflicted with lumpy jaw or is more common in people handling hay. Contagion from man to man has never been definitely established. The anaerobic organism of the type *Actinomyces bovis* has never been absolutely demonstrated in natural sources outside the human body.

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Wilkson, examining 10,000 tonsils, found bodies resembling those found in cases of actinomycosis in 177 cases. Weller, in an examination of 1,000 tonsils, found 47 out of 80 positive cases of tonsillar concretions of the crypts to be *Actinomyces*-like colonies of mixed mouth organisms. Lord found that patients with actinomycosis often gave a history of previous trouble with their teeth. He was also able to produce histologically identical lesions in 60 per cent of guinea pigs inoculated intraperitoneally with the contents of carious teeth of patients without actinomycosis. The lesions showed club-shaped *Actinomyces* granules.

Naeslund in 1925 was successful in isolating from the normal mouth anaerobic organisms morphologically and culturally like the Actinomyces of the Wolff-Israel type (*Actinomyces bovis*) and later produced characteristic lesions of actinomycosis in animals with these cultures.

PATHOLOGY

The pathology of actinomycosis is a granulomatous lesion associated with the production of fibrous and connective tissue. Multiple interconnected abscesses form, which rupture to the surface, producing multiple sinuses. The local lesions spread by direct extension through the connective tissue rather than by way of the lymphatics, although metastatic lesions appear in various parts of the body as the result of showers of infected emboli. The colonies of invading organisms are surrounded by mononuclear cells and an occassional giant cell. Numerous polymorphonuclear leukocytes are found in the necrotic areas and in the walls of the sinuses.

CLINICAL TYPES

The clinical types of the disease are: (1) cervicofacial, (2) abdominal, (3) thoracic, and (4) cutaneous or systemic. According to various references the cervicofacial region constitutes 50 to 60 per cent of the cases, the abdominal 20 per cent, the thoracic 15 per cent, and the cutaneous and systemic forms 5 per cent.

SYMPTOMATOLOGY

The clinical symptoms of actinomycosis vary somewhat, depending upon the region of the body involved.

In cervicofacial actinomycosis the infected area becomes indurated, extending beyond the area of inflammation and the overlying skin has a dark red or purplish color and a lumpy uneven surface. As the infection spreads, multiple sinus tracts appear and with internal extension the bones of the skull and even the meninges may be involved. Trismus develops when the muscles of mastication are involved. The lymph nodes of the neck are usually not involved and pain is not a prominent feature. The patient's general health remains good as long as the disease remains localized.

In thoracic actinomycosis, the symptoms are usually cough, sputum, increasing dyspnea, fever, weight loss, night sweats, pallor, and emaciation. The sputum may contain blood and fatal hemoptysis may occur. Pleural pains and effusions are not infrequent. After the disease reaches the pleura it usually involves the thoracic wall, extending through the subcutaneous tissue, and produces local areas of induration with suppurating foci and sinus formation. Cornell and Shookhoff,[8] quoting Ashford, state that in thoracic actinomycosis perforation of the chest wall occurs in more than 80 per cent of cases. From the thoracic foci there may be extension to the mediastinum, esophagus, and pericardium.

In abdominal actinomycosis, the earliest sign usually is the development of an indistinct, irregular mass in the ileocecal region, generally not painful and without characteristic features. It may or may not be accompanied by fever, chills, night sweats, intestinal colic, and vomiting. As the disease progresses there is usually involvement of the liver and spleen.

In the differential diagnosis the cervicofacial type must be differentiated from glanders, tularemia, tuberculosis, and osteomyelitis. The pulmonary form frequently simulates pulmonary abscess, tuberculosis, tularemia, and other mycotic infections. The abdominal form may be mistaken for appendicitis, amebiasis, typhoid fever, carcinoma of the intestines, tuberculosis, liver abscess, psoas abscess, and sarcoma of the retroperitoneal tissues or the iliac bone.

DIAGNOSIS

The diagnosis is based on the clinical syndrome, finding of the sulfur granules in the exudate or lesions, and cultivation of the organisms. Biopsies of the sinus tracts should be made when direct examination of the exudate fails to show granules. Pure cultures are readily obtained from material aspirated from unopened abscesses but isolation is difficult from draining sinuses because of the overgrowth from contaminating bacteria. The sulfur granules vary in size, having a radiating lobulated structure and are usually yellow in color. They are best observed with a low-power microscope lens but are occasionally large enough to be identified with the naked eye or a hand lens. The Gram-positive branched filaments can be demonstrated by crushing the granules between two slides and then staining with Gram's stain. These branched filaments make up the interior of the sulfur granule. The interior of the granule does not stand out sharply but the clubs at the periphery are refractile and appear as irregular lines marking the borders of the lobules.

The pus obtained should be washed with sterile normal salt solution, the granules then recovered with a bacteriologic loop, washed again in sterile normal salt solution, and then placed in thioglycollate media and incubated at 37° C. The inoculated tubes should be retained for four weeks before discarding. Colonies appear as fluffy masses of mycelium. An accessory means of diagnosis is the inoculation of guinea pigs with cultures of the exudate. Pure cultures of some strains produce a rapidly fatal disease in guinea pigs in 10 to 20 days.

PROGNOSIS

The prognosis is best in the localized skin and cervicofacial types and becomes worse with the thoracic, abdominal, generalized, and neurologic types.

Davis,[5] in 1941, reporting 46 cases of actinomycosis, stated that a follow-up study at the end of 10 years showed that of the 23 abdominal and thoracic cases, 22 were dead at the end of 14 months and the one living still had active lesions in the chest at the end of a year and a half. Fifteen of 23 cervical cases were followed. All but one was cured on the average of two and a half months after discharge from the hospital. One patient who had a persistent lesion on the cheek for 10 years finally died with generalized metastases.

Certainly with the advent of the sulfonamides and penicillin, the prognosis in the thoracic and abdominal cases should be better. Recent reports indicate that treatment with these drugs may be much more effective than with the usual remedies.

TREATMENT

Many drugs have been used in the treatment of actinomycosis and surgery has been advocated for many years.

Potassium iodide was first used in the treatment of lingual actinomycosis of cattle by Thomassen in 1885 and Nocard stimulated its introduction in the treatment of human actinomycosis by Itterson and Netter in the same year. At the present time it is thought that no indications for its use exist and *in vitro* studies reveal that Actinomyces grow luxuriantly on media containing 2 per cent potassium iodide.[19]

Thymol was first used in the treatment of cervicofacial actinomycosis by Myers and Theines in 1925. The drug was found to be effective against Actinomyces *in vitro* at a concentration of 1:1000. The organisms were killed following exposure to this concentration for one minute. Since 1925 various reports of the beneficial effect of thymol have appeared in the literature.[19]

The radiation therapy of actinomycosis was first introduced by Harsha in 1904. He used x-ray irradiation. Bevan in 1905 first used radium emanations in the treatment of the disease. Since then the efficacy of irradiation therapy in the circumscribed superficial forms of the disease is established without a doubt. It is ineffective in the widespread form of the disease.[19]

The principles for the use of surgery in the disease were first clearly stated by Waring in 1905.[19] Surgery has its use in the draining of collections of pus and the excision of hopelessly diseased and necrotic tissue. This is particularly applicable to the disease when it involves the internal female genitalia.

The sulfonamides were found to be effective agains the Actinomyces by clinical trial in 1937 and 1938.[19] *In vitro* studies in 1941 revealed that certain strains were almost completely inhibited when the media contained 50 mgm. per cent of the drug with sulfathiazole and sulfadiazine being more effective than sulfanilamide.[19] Pillsbury and Wassersug[16] reported successful treatment of a case of pulmonary actinomycosis with sulfonamides in conjunction with potassium iodide. A follow-up at the end of 10 months revealed no reactivation of the disease.

In 1941 *in vitro* studies revealed that penicillin was effective against a strain of *Actinomyces bovis.*[19] This organism was as sensitive to penicillin as the staphylococcus. Walker and Hamilton[2] reported the successful treatment of actinomycosis with penicillin. Two cases of soft tissue involvement, three cases of bone involvement, and one case of generalized systemic involvement were treated. Hendrickson and Lehman[17] reported two cases of proved cervicofacial actinomycosis treated successfully with penicillin and without the use of surgery. Roberts et al.[18] reported the successful treatment of a case of thoracic actinomycosis that was resistant to the sulfonamides. The first treatment consisted of 1,800,000 units by intramuscular drip over a two weeks' period. The fever recurred in three weeks and a second course of 5,600,000 units over a period of 28 days was given with good response. There was no follow-up.

Vaccines and copper sulfate have also been used in the treatment of the disease.

At present the sulfonamides, penicillin, x-ray irradiation, and surgery are the most effective therapeutic agents.

CASE REPORTS FROM LITERATURE

Bostroem isolated *Actinomyces graminis* from a case of human actinomycosis in 1891. Biggart in 1934 reported a fatal case of generalized actinomycosis due to *Actinomyces graminis.* Holt in 1941 reported a fatal brain abscess due to *Actinomyces graminis.*[1]

According to Beamer et al.[6] Alestra and Girolami in 1937 were the first to report a case of vegetative endocarditis due to Actinomyces (Nocardia, species not ascertained) diagnosed before death with blood cultures and proved by microscopic studies and cultures after death.[6] The second authenticated case of actinomycotic endocarditis was reported by Uhr in 1939.[6] The causitive organism was *Actinomyces bovis.*

Beamer et al.[6] in 1945 reported a case of vegetative endocarditis involving the aortic and mitral valve with the diagnosis being established at autopsy. The causative organism was *Actinomyces graminis.*

Lynch and Holt[1] in 1945 reported a case of pulmonary actinomycosis, due to *Actinomycosis graminis,* that eventually resulted in death due to extension to the pericardium giving rise to a subacute adhesive pericarditis, chronic myocarditis, and death.

Cornell and Shookhoff[8] in 1945 reported three new cases of actinomycosis of the heart and collected 65 others from the literature. They state that actinomycosis is the most common visceral mycotic infection in man. According to Kasper and Pinner, involvement of the heart and pericardium occurred in less than 2 per cent of 470 cases of actinomycosis.[8]

Rashbaum and McIntosh[9] state that actinomycotic infection of the internal genitalia of the female is uncommon and recovery is rare. Up until June, 1944, 85 cases of actinomycosis of the female pelvic organs had thus far been reported, only seven patients having survived. The authors reported the eighth case of actinomycosis of the internal female genitalia that responded over a long time to surgery and x-ray. A follow-up at the end of five and 10 years showed the patient to be well with no evidence of recurrence of abdominal or pelvic disease.

Smith[10] in 1943 reported a case of actinomycosis of the liver and lungs following the closure of a ruptured gastric ulcer. A sinus tract extended from the closure of the perforation over the anterior surface of the liver and thence along the thoracic wall, coming through the rectus muscle and skin to the surface.

Kasten[11] in 1945 reported a case of actinomycosis that had a primary granulomatous lesion of the right lung in the hilar region, with metastatic abscesses in the brain, around the thalamus and the fourth ventricle, accounting for a low fever and a slow pulse in the presence of a severe systemic infection.

Robinson[12] in 1944 reported a case of actinomycosis of the subcutaneous tissue of the left forearm, just below the elbow, following an accidental human bite. The lesion persisted for approximately three years and did not respond to treatment until adequate surgical incision was obtained.

Fuller and Wood[13] in 1945 reported a case of actinomycotic granuloma of the stomach. The stomach is a rare site for Actinomyces to cause a primary lesion, while intestinal actinomycosis is common. Fluoroscopic examination of their case revealed two ulcerative lesions of the stomach. Treatment was by sleeve resection, including the tumor masses, and a posterior gastrojejunostomy with a short loop. The pathologic diagnosis was actinomycosis. The patient was well 15 months after surgery, with no evidence of extension of the disease. The authors thought that this case was probably the only one on record of cured primary gastric actinomycosis.

Orr[14] in 1945 reported a case of actinomycoma of the third ventricle, probably primary, in which the cause of death was internal hydrocephalus due to a pedunculated ball-valve tumor causing intermittent obstruction of the aqueduct of Sylvius. He stated that descriptions of only four similar lesions have been found in the literature.

CASE REPORTS

1. The first case is that of a colored female, aged 35 years, who was admitted to the hospital on November 30, 1938, with the history of pain in the lower abdomen for four months. Treatment out of the hospital had been with oral medication and douches. The patient developed fever and later a mass appeared in the right lower quadrant of the abdomen. The essential finding on admission was a mass in the right inguinal region that was confluent with a mass in the right pelvic adnexa. An EPA of the chest and a barium enema were reported as negative. The mass was incised and drained and the diagnosis of actinomycosis was established by biopsy of the diseased tissue. Treatment was with x-ray irradiation and potassium iodide but improvement was only slight and the patient was discharged on March 9, 1939. From indirect sources it was learned that the patient expired of the disease. The patient's occupation was that of routine housework.

2. The second case is that of a white male, aged 17 years, who was admitted to the hospital on April 12, 1939. The past history revealed that the patient was subjected to an appendectomy in October, 1938. Following this an abscess developed in the right lumbar region. A barium enema was negative but an x-ray of the vertebra showed destructive changes in the third lumbar vertebra. Treatment was with x-ray irradiation, iodides, and sulfanilamide. The abscess was incised and drained. He was discharged on October 1, 1939, with no note as to the results. An indirect communication revealed that the patient expired. He had no occupation.

3. The third case is that of a colored male, aged 21 years, who was admitted to the hospital on June 13, 1939. Six months prior he noticed a small mass in the abdominal wall. On admission there was a mass from the ribs to the pelvis on the right side with draining sinuses in the right and left lower

quadrants of the abdominal wall. The EPA of the chest was negative. The diagnosis was made by biopsy of the diseased tissue. Treatment was with iodides and sulfanilamide and the patient was discharged on September 7, 1939, as improved. From indirect sources it was learned that the patient expired later. He worked on a farm.

4. The fourth case is that of a colored male, aged 35 years, who was admitted to the hospital on July 2, 1939, with the history of a lumbar abscess that had been present for six months and had been incised and drained before admission. Examination on admission revealed the pathology to be located in the lumbar region and the right lower quadrant of the abdomen. The diagnosis was made by biopsy of the diseased tissue. A barium enema and an x-ray of the vertebrae were reported as negative. Treatment was with iodides and x-ray irradiation. He was discharged August 4, 1939, with the note that the lesions were healed. He worked at a sawmill.

5. The fifth case is that of a colored female, aged 3 years, who was admitted to the hospital on November 9, 1940. The child had a lye stricture of the esophagus and developed, spontaneously, a right lumbar sinus. A barium enema was reported as negative. The diagnosis was established by biopsy of the sinus tract. Treatment was with iodides but the child expired on January 31, 1941. An autopsy was not performed.

6. The sixth case is that of a colored male, aged 70 years, who was seen in the Tumor Clinic in March, 1941. A mass at the angle of the right jaw had been present for two years. Following the extraction of a lower incisor, the mass began to drain a purulent exudate. At the time of examination there was a submandibular mass. The diagnosis was made by biopsy of the lesion. Treatment was with x-ray irradiation but there was no note as to the results.

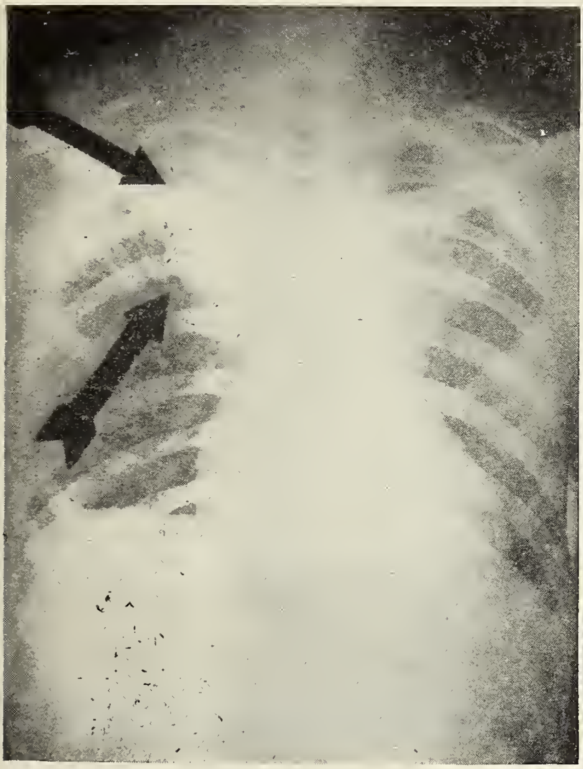
7. The seventh case is that of a colored male, aged 53 years, who was admitted to the hospital on February 18, 1942 with the history of an appendectomy five months prior to admission. Two months before admission he had an episode of pleurisy which was followed by dyspnea and swelling of the feet. Treatment was with sulfathiazole but he expired six days after admission on February 24, 1942. An autopsy revealed actinomycotic abscesses of the liver and peripancreatic tissues, chronic appendicitis, and pericarditis. In addition there was a bronchiogenic carcinoma with widespread metastases. He was an ordinary laborer.

8. The eighth case is that of a colored female, aged 27 years, who was admitted to the hospital for the first time on January 22, 1944. The diagnosis on this admission was pneumonia. During the stay in the hospital she developed pericardial and pleural effusions which necessitated pericardial aspiration and thoracentesis. The clinical course was very septic and she was discharged after three months, only to be readmitted later with a recurrent pleural effusion. She died soon after on June 5, 1944. Treatment was at irregular intervals with sulfathiazole and sulfadiazine. At autopsy there were lesions of the lungs, spleen, and liver. The causative organism was *Actinomyces asteroides.* She lived on a farm.

9. The ninth case is that of a white male, aged 28 years, whose last admission was on July 26, 1945. He had an appendectomy in 1943, following which he developed actinomycosis of the abdominal wall. This was incised and drained in 1943 and he was treated with sulfathiazole for one year. Drainage decreased but the lesions persisted and at a later date he was given 1,000,000 units of penicillin. On this last admission he was given 2,400,000 units of penicillin over a period of five days and was discharged July 31, 1945, as healed. In November, 1946 it was learned from an indirect source that the lesions were still draining but his general physical condition was good.

10. The tenth case is that of a colored male, aged 46 years, who was admitted to the hospital on December 18, 1945, with the history of a swollen knee for one year. The disease process began as a small pimple-like lesion that soon ulcerated and drained a sanguinopurulent material. Other sinus tracts soon appeared. For four weeks before admission the patient was bedridden. On admission the entire left knee was swollen, spindle-shaped, and had many granulating sinuses on the surface. The diagnosis was made by biopsy of the lesions. Treatment was with sulfadiazine, one gram every four hours, for a total dose of 257 grams. This was started on December 26, 1945, and stopped on February 6, 1946. In conjunction with this he received 4,260,000 units of penicillin from January 3, 1946, to February 8, 1946. Other medication consisted of potassium iodide for five days and vitamins. From January 7, 1946, to January 26, 1946, he received a total of 900 r. units to each of four ports. He was discharged on February 9, 1946, with the note that the lesions were nearly healed and drainage was no longer present. He has been seen in the Tumor Clinic five times since discharge and at the last visit on November 12, 1946, the skin was healed with no discharge present and no limitation of motion. The results are excellent.

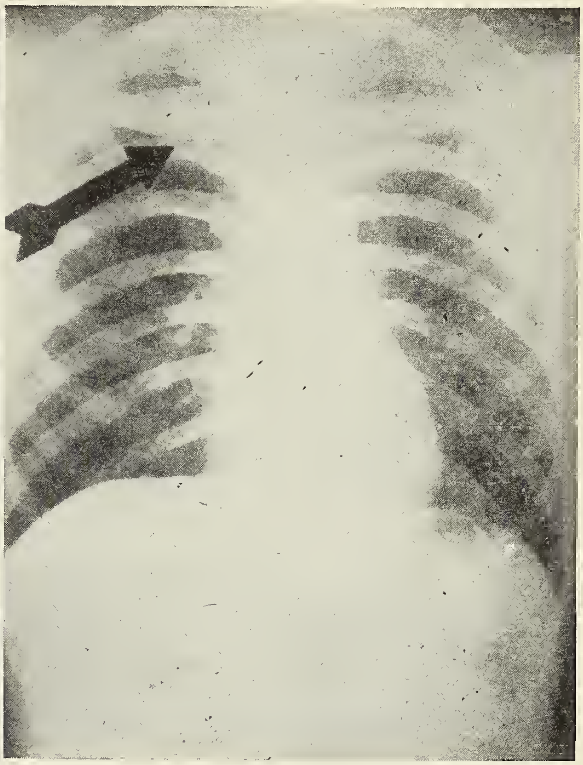
11. The eleventh case is that of a colored male, aged 20 years, first admitted in March, 1946, and treated for pneumonia with penicillin. The fever subsided and he was discharged on March 23, 1946. At this time he had evidence of a mass in the posterior mediastinum, the nature of which was not determined. He soon returned to the hospital with recurrence of fever, 99° to 103°, weight loss, pain in the back between the shoulders, and a history of slight hemoptysis on two occasions. On May 27, 1946, with local anaesthesia, an aspira-



LEGEND:

Fig. 1: An EPA of the chest on 3-13-46 showing a large ovoid mass of the right upper lung field with surrounding pneumonitis in the right lung and medial subapical portion of the left lung. The radiologist suggested a mediastinal mass or mediastinal fluid.

**Case 11*

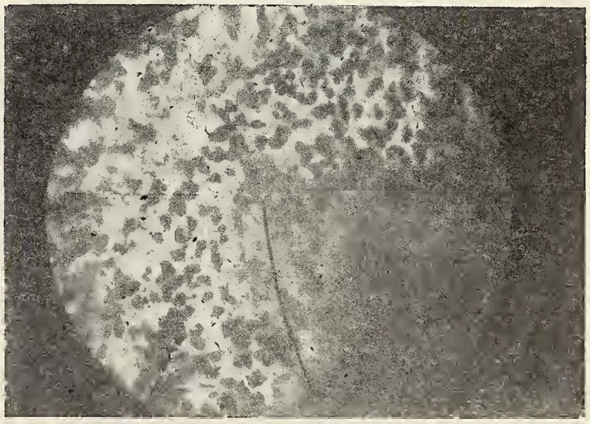


LEGEND:

Fig. 2: An EPA of the chest on 9-3-46 at the time of the return visit for a follow-up study which was reported by the radiologist as essentially negative.

**Case 11*

tion biopsy of the posterior superior mediastinum was done, the site of entrance being to the right of the spinal column in the third intercostal space. Sufficient material was obtained for a paraffin section. From May 21 through May 28, he received a total of 300 r. units to both a posterior and anterior port over the mediastinum, with no response. The diagnosis of actinomycosis was reported on May 31, 1946, and on this same date he was started on penicillin and sulfadiazine, which was continued until July 24, 1946. He received a total of 305 grams of sulfadiazine and 12,525,000 units of penicillin over this 56 day period. Seven days after starting chemotherapy he became afebrile and remained so until the time of discharge on July 25, 1946. He returned to the hospital on September 3, 1946, for a follow-up study. There was no evidence of reactivation of the disease on physical examination or by radiologic examination. He had gained weight, had been afebrile, and was able to work. He has not been seen since.



LEGEND:

Fig. 3: A photomicrograph of the paraffin section made from the material obtained by aspiration

biopsy of the mediastinum. It shows a microscopic sulfur granule in the lower field surrounded by numerous polymorphonuclear leukocytes.

**Case 11*

12. The twelfth case is that of a white male, aged 75 years, who was admitted to the hospital on May 13, 1946. Six weeks before, he had noticed a small mass on the posterolateral aspect of the thorax in the left infrascapular region. This continued to enlarge but was not painful or tender. Examination revealed a mass in the left infrascapular region about 15 x 10 x 3 centimeters in size. It had a bluish red color and was warmer than the surrounding tissue. There were no nodes in the axilla. An incisional biopsy was taken and the pathologic report of actinomycosis due to *Actinomyces bovis* was returned on June 7, 1946. Treatment consisted of 165 grams of sulfadiazine from May 13 to June 15, 1946, and 4.980,000 units of penicillin from May 22 to June 15, 1946. He was discharged June 19, 1946, with the note that the lesions were healed and the response to treatment was excellent. He has been seen in the Tumor Clinic on two occasions since his discharge, last on October 18, 1946, at which time the lesions were completely healed, skin soft and movable, and only two small scare remained at the site of the incisional biopsies. His general condition was excellent.

Since the final revision of this paper, two more cases of actinomycosis have been seen in this hospital. The first was that of a colored male, aged 43 years, who was admitted to the hospital on October 4, 1946 and expired on October 15, 1946. The main finding on physical examination was a markedly enlarged liver. Autopsy revealed actinomycosis of the liver with many gross abscesses and miliary lesions of the lung.

The other case was that of a white female, aged 30 years, who was admitted to the hospital in a delirious state with high fever and a draining sinus to the left of the vertebral column about the level of the tenth or eleventh dorsal vertebra. A biopsy of the sinus was taken and reported as actinomycosis. An x-ray of the dorsal vertebrae showed destruction of the left lateral border of the eighth dorsal vertebra and erosion of the vertebral end of the eighth and ninth ribs. She was placed in a body plaster jacket and started on penicillin and sulfadiazine. The response has been fair with the fever subsiding gradually but treatment is far from being complete at this time.

SUMMARY

Reference to the recent literature on actinomycosis has been made and 12 cases have been reported covering a period from November, 1938 to July, 1946. Since then two more cases of patients with actinomycosis have entered the hospital and they are briefly referred to.

The various types were as follows: genital, one; abdominal, six; cervicofacial, one; thoracoabdominal, one; subcutaneous, two; and thoracic, one. Of the six abdominal cases, two followed appendectomy. One of the subcutaneous cases involved the knee and the other invloved the back in the infrascapular region.

Of the two cases unofficially reported, the patients having entered the hospital after the final revision of this paper, one was a case of osseous involvement and the other was a thoracoabdominal type.

Of the 12 cases officially reported, there were six deaths, a mortality of 50 per cent.

The last three cases reported (cases 10, 11, and 12) certainly reveal the efficacy of the sulfonamides and penicillin in the treatment of the disease and illustrate the necessity of establishing the proper diagnosis in any suspected mycotic infection.

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MULTIPLE OPERATIVE PROCEDURES IN A CASE OF VON RECKLINGHAUSEN'S DISEASE

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NEW ORLEANS

Von Recklinghausen's disease is always of interest to the clinician because of its rare occurrence, familial characteristics, progressive course, and protean manifestations. Typically, it is characterized by multiple cutaneous tumors, pigmentary anomalies, and tumors of the peripheral, central and sympathetic nervous systems. Manifestations may vary from a few skin nodules and café-au-lait spots to evidence of widespread involvement of the sheaths of the nervous system. The skin may exhibit predominant, minimal or no manifestations of the disease.[1] Familial incidence may be striking[2, 3] or of little degree. Pathologic findings may vary considerably from case to case and even within a single tumor.[4]

Involvement of the central nervous system[5] and of the peripheral nerves[6] is frequently of surgical importance. Jones and Hart[7] classified patients in whom surgical complications develop during the course of von Recklinghausen's disease into three main groups. The largest group is composed of persons in whom neurofibromas appear in such a location that pressure from the tumor affects the function of one or more vital structures. The second group consists of those patients in whom a malignancy develops in the course of the disease. The third group comprises those patients having a hemorrhage into a large pachydermatocele. The following case, which falls into group one, is presented because it illustrates one course which the disease may take and also because multiple operative procedures were necessary to maintain the patient's health.

CASE REPORT

A. L. T., a 55 year old, obese, Italian woman, was admitted to Charity Hospital on July 31, 1946, with a chief complaint of acute, agonizing, low back pain of two weeks' duration. This was her fourteenth hospital admission, her tenth for complaints referable to the course of von Recklinghausen's disease.

The patient was first seen September 11, 1930, and followed in the out patient department until December 31, 1931. Her history dated from approximately two years before admission when, after several days of dizziness and weakness, she awoke one morning to find that she could not move the left side of her face or use her left leg properly. Permanent residuals from the attack were loss of facial movements on the left and some slight weakness and impairment of control on the left lower extremity. The cause of these symptoms has never been explained.

Examinations during 1930 and 1931 showed slight blurring of the optic disks, left facial paralysis of the peripheral type, left nerve deafness, enlargement of the left internal auditory canal and destruction of the apex of the petrous portion of the left temporal bone as seen in the roentgenograms. A presumptive diagnosis of a tumor of the left cerebellopontine angle was made but the patient refused to have an operation.

In July, 1933, the patient still presented signs and symptoms of a cerebellopontine angle tumor. The reason for the return visit was a freely movable, painless mass on the upper posterior aspect of the left thigh. To her knowledge it had been present for a year, growing from the size of a hen's egg to that of her fist. During that year she had noticed, on squatting, stooping, or sitting, numbness in her leg which became progressively more frequent and severe. Removal of the tumor in the thigh relieved the symptoms. No surgical or pathologic report is available.

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From October to November, 1937, the patient was treated on the gynecologic service because of vaginal bleeding. Examination disclosed a hypertrophied and lacerated cervix, relaxed perineum, and second degree uterine retroversion. The previously noted cranial nerve palsies were unchanged. A roentgenogram at this time showed the defects in the petrous portion of the temporal bone noted in 1933. On one of these visits a small mass was complained of on the medial aspect of the right arm about two inches below the axilla. It was firm, oval, and attached to the underlying structures. Pressure on it produced tingling in the third, fourth and fifth fingers. In a similar position on the left arm another but asymptomatic subcutaneous mass was found. The patient refused to be treated for these lesions.

The patient was again admitted to the surgical service in January, 1939, complaining of headache, "scratchy" sensations in both ears, nausea and vomiting. On examination, facial paralysis and nerve deafness were present on the left side, as previously noted. The visual fields were normal. The eyegrounds showed definite blurring of the nasal margins of both disks, with a beginning choked disk on the left. The tumors previously noted on the arms were unchanged. The epigastrium was tender and one observer felt an ill defined mass there. Laboratory procedures including barium studies of the intestinal tract, Wassermann test, gastric analysis, and stool examination revealed findings within normal limits. A caloric test showed no reaction of the left vestibular apparatus. Roentgenograms of the skull on February 14, 1939, showed erosion of one side of the dorsum sellae, of the basilar process of the occipital bone and also of the petrous portion of the left temporal bone. The headache, nausea and vomiting gradually subsided and the patient was discharged to return at a later date for surgical exploration of the cerebellopontine angle.

The patient was readmitted on May 9, for craniotomy. Her condition had not changed appreciably. Preoperatively, a diagnosis of neurofibroma involving the seventh and eighth cranial nerves on the left side was made. The operation was performed under local anesthesia on May 13, 1939, by one of us (D. H. E.).

The cerebellar hemisphere, which appeared normal, was retracted without difficulty to expose extensive adhesions involving the seventh, eighth, ninth, tenth, and eleventh cranial nerves. There was no tumor in the angle. Nothing further was done. The postoperative course was uneventful and the patient was discharged on the ninth postoperative day.

The patient was readmitted February 14, 1940. Since discharge in May, 1939, she had not experienced headache or vomiting. She was not conscious of the pulsating defect in the left suboccipital region. About two weeks before admission she began having an aching pain on the right side in the supraclavicular region, the shoulder, the inner side of the forearm, and the little finger. Pressure applied to the mass on the medial aspect of the right arm produced numbness in the third, fourth, and fifth fingers.

On February 27, 1940, under local anesthesia Dr. Mims Gage explored the mass on the right arm. A fusiform tumor, which measured 3 cm. x 2 cm., was found on the ulnar nerve. The tumor was cystic. It was completely removed without interrupting the continuity of the nerve. The pathologic diagnosis was neurofibroma.

In February, 1942, the patient came in because of pain behind the left knee traveling up the posterior aspect of the thigh to the buttock. The pain was made worse by bending forward. There was a decrease in the left Achilles and patellar reflexes. There was numbness and tingling over the ulnar distribution on the right hand with atrophy of the hypothenar eminence. The mass on the left arm was unchanged. Facial paralysis and nerve deafness on the left side were still present. The patient was allowed to go home since her difficulties appeared minimal.

The patient was readmitted a month later on May 16, 1942, because the pain in her back and leg had become worse. She had been confined to bed for three days before admission with cramping pain in the calf muscles of the left leg, severe backache, intermittent shooting pains originating in the back and left buttock and radiating down the left leg, and severe, lancinating pain in the lower part of the back on extension of the left leg.

On examination, the left lower extremity was weaker than the right; there was exquisite tenderness in the sacro-iliac region and over the sacrum; pain on extension of the left leg was referred to the left buttock and lower part of the back; and loss of position sense in the toes and absent Achilles tendon reflexes were found bilaterally. A diagnosis of neurofibroma of the cauda equina was made.

On April 29, 1942 under local anesthesia one of us (D.H.E.) removed the spinous processes and laminas of the lower three lumbar vertebrae. The dura was opened to expose an arachnoidal cyst, which completely filled the dural sac. The wall of the cyst, which was thin and transparent, collapsed when it was opened. At the cephalad pole of the cyst was a neurofibroma 1 cm. in diameter attached to a nerve root. Apparently, the cyst was causing the sciatic pain by pushing the tumor in a cephalad direction. This tumor and five or six smaller ones attached to various roots were removed and the dura was closed. These were diagnosed pathologically as neurofibromas. The pain was completely relieved and the patient was discharged on the sixteenth postoperative day.

In November, 1942, the patient was readmitted to the hospital with the same symptoms as on her previous admission but in the other leg. These had appeared one month after discharge. Myelography with lipiodol showed a filling defect at the level of the fourth and fifth lumbar vertebrae. A diagnosis of neurofibromas of the cauda equina was made. Operation was deferred at the patient's request.

In January, 1943, the patient returned to the hospital. Since discharge she had had continuous cramp-like pain in the lower lumbar region and right thigh. The pain became worse on coughing, sneezing or sitting down and radiated from the lower lumbar region on the right side down the posterior aspect of the right thigh.

Operation was performed by Dr. J. A. Colclough on January 21, 1943. Under spinal anesthesia the previous laminectomy incision was opened. The cauda equina was found to be compressed and adherent to two neurofibromas. Because of the adhesions it was not possible to remove these tumors but the first sacral root was identified extradurally and severed. No attempt was made to close the dura. The patient was relieved of the sciatic pain and went home on the tenth postoperative day.

Nothing was heard from the patient until the last admission, July 31, 1946. The patient could not give any competent evidence concerning the presence of signs of von Recklinghausen's disease in her parents, brothers and sisters, or others beyond the present generation. She has three daughters and one son. The son has café-au-lait spots on his shoulders and forehead. One daughter had a left radial nerve tumor removed from her forearm in 1933. The pathologic diagnosis was "fibroblastoma of sheath showing some myxomatous degenerative changes." The other two daughters show no sign of the disease.

Since her last operation on January 21, 1943, the patient had remained at home doing some of her housework. During the last two years attacks of back pain had steadily become more severe. For two weeks before admission the patient had been forced to remain in bed because of extreme pain in the lower part of the back which radiated down the posterior surface of the thighs and legs to the ankles. After the pain in the leg had become severe, the back at the upper end of the laminectomy scar became exquisitely tender. Since her last operation the bowels could be evacuated only by the use of cathartics or enemas. Self-catheterization was used for two weeks. She had taken pantopon by hypodermic several times a day for twenty-four months.

Examination on this final admission disclosed a rather obese, stocky woman lying quietly in bed. Pigmented moles and freckles were widely distributed over her body. The cranial nerves were normal except for the incomplete facial paralysis and nerve deafness on the left side. The left suboccipital decompression (1939) showed pulsations and did not bulge. The scars of the previous operation on the left thigh (1933) and right ulnar nerve (1940) were well healed and the tumors had not recurred. A soft, lobulated mass was present on the medial side of the left arm about 5 cm. below the axilla. It was firmly attached to the underlying structures and was not sensitive. A long midline lumbar scar was present, the site of previous operations (1942, 1943). The slightest pressure over the upper end of the lumbar scar was exquisitely painful and caused the pain to radiate down the back of the left leg. Achilles and patellar reflexes were absent bilaterally. Straight leg raising produced such severe pain in the back that the patient became uncooperative. The patient was only partially relieved by 1 grain doses of morphine at short intervals.

It was obvious that something had to be done surgically to control the pain but it seemed desirable to postpone bilateral chordotomy. Operation was performed by one of us (D.H.E.) on August 8, 1946. Under endotracheal ether anesthesia the dural sac was exposed from the eleventh dorsal vertebra to the sacrum. Three large tumors were found, two of which compressed the lower end of the spinal cord. A nerve entered and left each of these tumors. The three large tumors were excised. Multiple small tumors were found attached to various nerves. These were removed as well.

The pathologic report was neurofibroma with focal degeneration, hemosiderin deposits, venous thrombosis, and pleomorphic cells suggesting rapid growth.

Postoperatively, severe cystitis developed and the patient's course was stormy. The wound healed primarily and by the thirteenth opstoperative day when the patient was discharged, urination was voluntary and controlled. The motor and sensory losses were surprisingly small and pain was considerably decreased. In view of her known addiction to pantopon she was given permission to continue use.

The patient was examined at her home on January 10, 1947, and found to be in reasonably good health and spirits. She was able to get around the house and do light work. Her chief complaint was constipation; bladder control was said to be normal. Pantopon by mouth in diminishing doses had been substituted for hypodermic administration.

DISCUSSION

In 1932 this patient first noticed a tumor on her left thigh. She was then 41 years old, well beyond the usual time of onset of von Recklinghausen's disease. Since then she has undergone removal of intraspinal and peripheral neurofibromas on four occasions. Though late in appearing the disease has been progressive.

According to Hosoi[8] malignant transformation takes place in about 13 per cent of all cases of von Recklinghausen's disease. For a complete study of this problem his paper should be consulted. He describes two cases in which malignant changes took place about one year after operative intervention and reviews this phase of the literature. It is suggested that tumors of the deep peripheral nerves should be treated by wide excision but he points out that if sarcomatous change and recurrence take place, repeated operative intervention is of no avail. It is also interesting that after removal of one tumor, another tumor on the body may undergo sarcomatous degeneration. Metastasis in these cases is usually late. A case in point was reported by Jelsma[9] in discussing the diagnosis of hourglass tumors of the cervical spine. This tumor was a neurofibroma which underwent operative intervention on four occasions and on the fifth malignant change had occurred. In the case reported by us there has been no recurrence at the site of removal of the tumor of the right ulnar nerve or of the left thigh (probably sciatic) and the only other known peripheral tumor has remained unchanged.

Our patient had operations for cauda equina neurofibromas in 1942, 1943 and 1946. On the first occasion a cyst and several small neurofibromas were removed. At the second operation a decompressive procedure was done. On the last occasion three large and multiple small neurofibromas were removed. Many pleomorphic cells which were not present previously were seen in sections of the most recent tumor. This suggested to the pathologist an indication of rapid growth. Malignant transformation in some of the remaining tumors may eventually take place.

Because of the frequency of malignant transformation in von Recklinghausen's disease and its tendency to follow surgical intervention, operations should not be needlessly performed. Indications for operation have been pointed out. In our case operation was undertaken on five occasions when pain was severe or when life was threatened. Exploration of the left eighth cranial nerve failed to disclose an acoustic neurofibroma and the roentgenographic changes in bone are unexplained but there had occurred some pathologic process which simulated a tumor of the eighth nerve. The recognized frequency of acoustic nerve tumors in multiple neurofibromatosis plus the left nerve deafness led to the exploration. As yet no malignant change has appeared in this case and the patient has benefitted from each surgical approach to her troubles.

SUMMARY

A case of a 55 year old Italian woman who has been operated on six times as a consequence of multiple neurofibromatosis is presented. The tumor tissue from the last operation showed some indication of rapid growth.

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TRAUMATIC RUPTURE OF THE PANCREAS

CASE REPORT

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Traumatic pancreatic rupture, a rare entity, was first described by Travers[1] (1827) in association with injury to other intra-abdominal viscera and by Kulenkampff[2] (1882) as an occurrence with the pancreas alone damaged. The latter is more unusual; solitary involvement represented 26.6 per cent of traumatic ruptures in Von Garre's[3] study, Mocquot and Costantini[4] found only 30 such instances in their review of the literature. A decade of admissions (1935-1945) at Southern Baptist Hospital and Touro Infirmary, 314,005 admissions, revealed only the incidence of pancreatic rupture that sponsored this case report.

Etiologically the three possibilities are external trauma, penetrating wounds and operative procedures. Any external trauma compressing the abdomen may be causative, a wide variety of accidents have been reported; Keynes[5] recently added a war-time source, the compressive force of an underwater blast. Stern[6], Schmeidin and Sebening[7] have thoroughly considered injuries arising from surgery. Penetrating wounds frequently show concomitant injury to other intra-abdominal viscera. Neither the operative nor the penetrating variety, in the obviousness of their origin, have the diagnostic interest of the external type so often concealed by the absence of external evidence of abdominal injury.

The manifestations of traumatic pancreatic rupture when not accompanied by concomitant visceral injury, vary from the typical acute abdomen, shock, cyanosis and skin changes of a pancreatic catastrophe to a mild gradually developing, often intermittent, peritoneal reaction. In severe injury the acute clinical picture developing subsequent to trauma, in the absence of other demonstrable associated visceral injury, may stimulate the diagnosis. A gradually appearing diffuse but mild peritoneal reaction, at times intermittent and especially accentuated postprandially, which localizes after an interval of days to weeks in a palpable epigastric mass, is typical of milder pancreatic ruptures which eventuate in pancreatic cysts. Gravitation may cause appendiceal localization to confuse the picture. The peritoneal reaction does not usually excite the febrile or leukocytic response of an inflammatory peritonitis nor is the ileus as severe.

Cognizance of the possibility, with elimination of other visceral or spinal injury as etiologic, will incite conclusions from such confirmatory laboratory findings as glycosuria, hyperglycemia and markedly elevated serum amylase that pancreatic injury has occurred. Peritoneal aspiration of fluid for tryptic reaction has been advocated.

Operative intervention is obviously essential in the majority of instances as an emergency measure, in some it is a diagnostic necessity, in an elective management it is resported to for correction of complications. Excellent surgical references cover the issue.[8, 13, 16]

Roentgen therapy[14, 15, 16, 17] has a limited and controversial application alone or in conjunction with surgery; because of the severity of the issue and the occasional definite efficacy of this therapy, it should be considered in all instances.

Individualized instances are managed conservatively. Medically, aside from analgesics, treatment of shock and maintenance of fluid balance there is much that can be done either as an adjunct to surgery or where operation is not elected. Since any oral intake stimulates pancreatic secretion, all oral nourishment should be withheld and parenteral fluids, proteins, plasma and blood should maintain the patient as long as the serum amalyze level remains elevated or fluctuates. Continuous duodenal suction besides combating ileus will reduce the stimulation of content. When feedings are resumed it will probably be best to resort to duodenal intubation gavage as suggested by Comfort and Priestley.[17] When oral feedings are started, the investigations of Wohlgemuth[18, 19, 20] and Pavlov[21] are still substantiated that a low carbohydrate diet rich in alkaline foods, large doses of sodium bicarbonate and belladonna will be best in controlling pancreatic secretion since the hormonal secretory stimulation arises from acid chyme flowing into the duodenum. A "mixed diet" relatively low in fat was theorized best by McCaughan and Purcell.[16]

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Ephedrine and epinephrine, pilocarpine, physistigmine, mecholyl, and atropine have all been mentioned as adjunctive therapy to surpass pancreatic secretion.[16] They, like nitroglycerine, probably act through depression of the vagus innervation of the pancreas.

A study of the situation indicates the prognosis generally to be poor. This, of course, varies in direct relation to the severity of the pancreatic injury. Mild cases may subside spontaneously or with reasonable conservatism, resultant pancreatic cysts have even resolved under conservative management. The more severe cases require operation, carry a high operative mobidity and mortality and are frequently multiply complicated by any of the following: pancreatic abscess, fistula, cysts, diabetes mellitus and chronic pancreatitis. The following case report is therefore of interest because of the fortunately favorable response to combined medical, roentgen and surgical management.

CASE REPORT

R. Y. T., a 15 year old white male, was admitted to Southern Baptist Hospital September 23, 1945. Five days prior to hospitalization he fell, forcefully striking his abdomen against the foot of a fellow player. He was unconscious and in shock for about 60 minutes; on recovery, he complained of severe generalized abdominal pain, became nauseated and vomited. He was maintained by parenteral fluids at home but showed no improvement.

Physically the patient was acutely ill, afebrile and free from shock. Localized pain, tenderness and muscular rigidity were present over the upper right rectus muscle. There were no abnormal palpable masses. The only positive laboratory finding was a leukocytosis with 90 per cent neutrophiles. The stools were normal. There was no glycosuria. Blood dextrose was 81 milligrams per cent. Roentgen study revealed no abnormality.

The patient's seven day hospital stay was uneventful except for a low grade intermittent fever. No specific therapy was given. A diagnosis of traumatic myositis was presumed.

With resumption of a normal diet, upon discharge from the hospital, the patient had recurrent, progressively severe, diffuse abdominal pain and tenderness. Weight loss of 15 pounds and profound asthenia indicated readmission on October 16, 1945.

Readmission examination revealed pyrexia (100.4°), generalized abdominal pain and tenderness with rigidity over the left upper quadrant. No masses were palpable. Leukocytosis of 20,000 with neutrophilia and eosinophilia, albuminuria and mild glycosuria were the significant laboratory findings. Hookworm ova were found in a routine stool examination and were considered coincidental; the stool was otherwise normal. Chest roentgenologic findings were normal. A flat plate of the abdomen revealed "distended small bowel suggesting partial intestinal obstruction." Intravenous pyelography was normal. A subsequent gastrointestinal series with small bowel study was reported negative.

The hookworm infestation was eradicated by hexylresorcinol. There was no febrile response to penicillin.

Operation was elected by Dr. T. B. Sellers with a preoperatitve diagnosis of traumatic injury to an unknown abdominal viscus. Extensive periduodenal and lesser sac adhesions were encountered with a fluctuant cystic mass occupying the mid portion of the body of the pancreas. Thirty c. c. of serosanginous fluid was aspirated and a drain inserted with peritoneal suturing to isolate the catheter. The peritoneal reaction was that of a chemical peritonitis; 380 c. c. of free peritoneal fluid was aspirated. The other abdominal viscera showed no abnormality. Analysis of the aspirated peritoneal fluid revealed more than 200 amylase units per cubic centimeter.

Postoperative management included repeated blood transfusions, plasma and adequate fluids parenterally. All feedings were parenteral. Atropine and aluminum hydroxide were given. Subsequently, when the serum amylase stabilized, he was given raw pancreas orally as a supplement to vitamins and a low fat diet. Deep roentgen therapy was instituted on the tenth postoperative day.

Convalescence was slow but uneventful, drainage subsided after 26 days and the patient was discharged on the thirty-third postoperative day and continued asymptomatic until December 9, 1945 when he developed nausea, emesis and right lower abdominal pain. This condition persisted with localization of a mass in the appendiceal area indicating rehospitalization on December 21. Exploration by Dr. J. T. Sanders revealed a cystic mass in the appendiceal area containing 420 cubic centimeters of straw colored fluid. Marsupialization was again applied. The medical management was that of the previous postoperative period and again a series of deep roentgen treatments were given. Drainage was completed in six days and the patient was discharged on the tenth postoperative day (December 30, 1945).

The patient remained asymptomatic. Reexamination on May 2, 1946, at which time he reported a weight gain of 21 pounds, revealed a palpable epigastric mass presumably a pancreatic cyst. On examination July 1, 1946 this mass had completely subsided. He continues asymptomatic with normal blood amylase, no glycosuria or hyperglycemia and all stool studies show a normal fat content.

CONCLUSION

The rarity of traumatic pancreatic rupture of the external variety without associated injury to any other viscus justifies this case report. Pancreatic cysts are the most frequent cause of fistulas. It is therefore fortunate that marsupialization in two instances did not result in this complication. We may assume that careful medical management and adjunctive roentgen therapy by suppressing pancreatic secretion may have influenced the favorable result.

A hint toward conservatism is gathered in the confirmation that pancreatic cysts may subside spontaneously.

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AN EVALUATION OF THE PSYCHOGENIC FACTOR IN PEPTIC ULCER*

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In a scientific approach to any disease process, a factor of fundamental importance is exact knowledge concerning its mode of causation. Yet notwithstanding the unprecedented progress of science in general, and medicine in particular during the last half century, one still witnesses the paradox of incomplete knowledge of the pathogenesis of numerous common disease entities. The inclusion of chronic gastroduodenal ulcer in this category is unfortunate but necessary. We might well say that the etiology of this condition has become a perennial problem and one has but to consult the voluminous literature on this subject to recognize how true this is. Aside from academic considerations, a continued interest in problems concerned with the etiology and pathogenesis of chronic gastroduodenal or "peptic ulcer" is justified by one fact alone, the great prevalence of this condition affecting as it does the health and efficiency of so many of the adult members of every community. Moreover, statistics accumulated from all over the world reveal an alarming increase, both relative and absolute, in the incidence of peptic ulcer in both civilians and military personnel. The British have reported an incidence of 35 to 55 per cent of ulcer in their gastrointestinal cases.[1] In a consecutive series of 15,000 patients with chronic dyspepsia examined in the Mayo Clinic, 15.5 per cent had deformities which were interpreted as being the result of peptic ulcer.[2]

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We might well ask ourselves why, in spite of extensive investigations over a long period of time, the problem of the etiology, pathogenesis and treatment of peptic ulcer still remains a very real one. In this connection, it is well to remember that, in the first place, peptic ulcer is a disease peculiar to man. Secondly, the production in laboratory animals of the chronic lesion is only possible after extensive operations on the viscera, producing such profound alterations in function that it is difficult to conceive of analogous changes on the intact human stomach or duodenum. Thirdly, if we are to accept the view that psychogenic or neurogenic factors play prominent roles in the etiology of this condition, experimental verification proves extremely difficult. However, it is to be acknowledged that experimental research has provided a great deal of information concerning the pathogenesis of the chronic lesion which can be correlated with many of the pathologic facts and clinical observations.

In examining the literature, one is impressed with the variety of conflicting views represented by numerous theories regarding the nature of peptic ulcer.

A recent survey by Cook and Fuller[3] revealed some eighteen theories still rather widely supported. These included chemical, infectious, constitutional type, mechanical, gastritis, deficiency, endocrine, vascular, mucosal irritant, traumatic, neurogenic, deranged motility, embrological anomalies, metabolic disturbances, allergy, dietary factors.

Fortunately, most of the important contributions of the past few years are in agreement on one point, that peptic ulcer cannot be explained by the action of any single agent but is the result of the actions and interactions of several agents. Just where the psychogenic factor fits into the picture is the subject of this paper.

Cushing,[4] in an excellent paper, observed that all clinicians are familiar with certain facts.

1. That highly strung persons are particularly susceptible to nervous indigestion and associated ulcer.

2. That ulcers become symptomatically quiescent or even tend to heal when patients are put mentally and physically at rest.

3. The symptoms are prone to recur as soon as the victim of the disorder resumes his former tasks and responsibilities. He concluded his paper with the suggestion that highly strung vagotonic persons subjected to emotional stress with concomitant factors such as irregular meals and the excessive use of tobacco, are prone to have chronic digestive disturbances often resulting in ulcer. Cushing was stimulated to make this review after his observation of three cases of early fatality from peptic ulcer following operations for cerebellar tumor. What is perhaps most noteworthy in the article is its pointing out the inadequacy of the term "neurogenic" as an ultimate etiological factor since neural mechanisms in this case serve as a pathway for the emotions, especially repressed emotions and consequently "psychogenic" would be the better term. It was Cushing's suggestion that the influences arising in the parasympathetic center in the hypothalamus and conveyed along the vagus nerve, are responsible for changes in the gastric mucosa which lead to the development of ulcer.

The experimental investigations of Beattie,[5] and of Keller and his associates[6] support this conception. By stimulation of the hypothalamus in the region of the tuber cinereum Beattie produced areas of hyperemia and small erosions in the lesser curvature of the gastric mucosa. After section of the vagi these effects could not be obtained. Keller has reported ulcerations of the stomach and proximal duodenum with hemorrhage, following lesions of the hypothalamus. The injection of pilocarpine (a parasympathetic stimulant) into the third ventricle or continued stimulation of the vagus nerve will produce hyperemia and erosions of the gastric mucosa.[7]

If we accept the fact of a relationship between the hypothalamus and the peptic ulcer what evidence have we concerning the relationship of emotional states and the hypothalamus? Although recent experimental and clinical investigations have revealed the hypothalamus as a structure of great physiologic importance, our knowledge of the functions of this part of the cerebrum remains incomplete. However, it is generally conceded that the mechanisms controlling certain primitive reactions (visceral and somatic) are contained in the hypothalamus. These reactions are associated in animals with defense or attack and in man with emotional states such as fear and anger. Goltz[8] was the first to demonstrate that the reactions which usually accompany displeasure and anger are more readily invoked in the decorticated animal than in the normal one. In a classical experiment, the hemispheres and a large part of the thalamus were removed from the dog. This operation greatly altered the disposition of the animal. The least provocation resulted in barking, growling, baring the teeth or snapping. Cannon and Britton[9] produced similar states in cats by means of decortication. These observers applied the term "sham rage" to the lashing of the tail, erection of the hairs, protrusion of the claws, dilatation of the pupils, sweating, struggling and biting, and greatly increased respiratory rate and blood pressure following recovery of the animals from the anesthetic. If we examine the results of decortication experiments as a whole, we are led to the conclusion that normally, the activities of the hypothalamus are under inhibitory influences from the cerebral cortex and "sham rage" would then represent the release of these primitive subcortical centers from higher control. It is well known that among the effects of disorders in the hypothalamic region in human beings are emotional manifestations, such as laughing, crying, or states analogous to "sham rage" in animals. In addition, are a variety of phenomena attributable to sympathetic or parasympathetic stimulation.

We now have some approach to the problem of peptic ulcer from the psychogenic standpoint in that we can visualize a mechanism mediated by tangible anatomic structures having its stimulation in the psyche. Whether the local problem in the stomach is due to hypersecretory, hypermotile, vasospastic, or any other of the suggested local mechanisms is not within the scope of this paper. It should be mentioned, however, that a large amount of noteworthy work has been contributed regarding the changes in the gastric mucosa mediated through the nervous system.

Von Bergmann's work is discussed in an excellent article by Eusterman.[10] He (V. Bergman) has expressed the belief that disharmony between the vagus and splanchnic systems (parasympathetic and sympathetic) produced localized or generalized spasm in the muscles of the stomach, duodenum, or terminal blood vessels. As a result of such dysfunction, areas of submucosal ischemia or decreased resistance developed, and resulted eventually in the formation of mucosal erosions through action of the gastric juices. Recently, Von Bergmann has reported gastroscopic observations of the development of such lesions from the initial stages to the advanced ulcus collosum.

Kuntz,[11] in his textbook on the autonomic nervous system, mentions that the association of parasympathetic hypertonus with gastric and duodenal ulcers has long been recognized. He cites important clinical, pathologic, and experimental observations supporting his contention.

More recent knowledge concerning the chemical mediation of autonomic nerve impulses has brought forth etiologic concepts based on the activity of the parasympathomimetic factor, acetylcholine. Of interest in this field are the observations of Necheles.[12]

It is now a rather well-established fact recognized by even the most organically minded clinicians that the alimentary tract is used with definite predilection by the psychic apparatus in the relief of various tensions with strong emotional content. However, it is unfortunate that in medicine there still remains the attitude that when one has carefully eliminated organic origins, especially in the so-called "nervous" patient, one is justified in the assumption of psychic factors. There then commonly follows an indulgence in rather vague, well-used terms such as "nervous exhaustion" or "unstable personality" or in equally indefinite technical terms such as "neurasthenia", "psychasthenia", "psychopathy" and a host of like expressions. Further, many physicians after satisfying themselves that the patient is overburdened in some fashion by the emotional strain of worries, fears, and anxiety feel qualified to offer therapeutic advice. Moreover, if one consults the literature, he will find that in general even those authors who attribute the greatest importance to psychic factors show little inclination to delve into the exact nature of the patient's psychologic situation. I feel, therefore, that a presentation of the observations of workers concerned with the more precise mechanisms involved in the etiology and pathogenesis is within the scope of this paper since it is here that one approaches the basis of the problem and can subsequently develop an intelligent and comprehensive attitude toward it.

In an excellent article on the emotional component of the ulcer susceptible constitution, Draper[13] describes evidence of autonomic irritability in a large number of his patients. These include easily sweating palms, tendency to wide palpebral fissures, slight sinus arrhythmias, and prompt slowing of the heart rate by pressure over the carotid sinus and deep breath holding. He notes that on admission to the ward most ulcer patients display an average or elevated pulse rate but after a day or so this frequently falls to a slower than average basic count. A large majority of his cases had good to superior intellects and possessed swift and intense emotional responsiveness. These patients were often conscientious to the extreme, high principled and striving. In an analysis of eighty subjects, Draper found a distinct tendency to regression (that is, an unconscious effort to regress toward infancy.) This finding has been described by many other investigators and will be discussed in greater detail later.

A recent and unique series of experimental studies made by Wolf and Wolff[14] on a man of 56 who suffered an occlusion of the esophagus at the age of nine and who has fed himself ever since through a surgically produced gastric fistula has led additional support to the concept of psychogenic factors in peptic ulcer.

Their conclusions are stated as follows:

1. Emotions such as fear and sadness, which are involved in a feeling of withdrawal, were accompanied by pallor of the gastric mucosa and by inhibition of secretions and contractions.

2. Emotional conflicts involving hostility, anxiety and resentment were accompanied by accelerated acid secretions, hypermotility, hyperemia and engorgement of the gastric mucosa resembling "hypertrophic gastritis." This series of events was the one most commonly observed in their subject. It was associated with gastrointestinal complaints of the nature of heartburn and abdominal pain.

3. Intense sustained anxiety, hostility and resentment were found to be accompanied by severe and prolonged engorgement, hypermotility and hypersecretion in the stomach. In this state, mucosal erosions and hemorrhages were readily induced, even by the most trifling traumas, and frequently bleeding points appeared spontaneously as a result of vigorous contraction of the stomach wall.

4. Contact of acid gastric juice with such a small eroded surface in the mucosa resulted in accelerated secretions of acid and further engorgement of the whole mucosa. Prolonged exposure of such a lesion to acid gastric juice resulted in the formation of chronic ulcer.

5. The lining of the stomach was found to be protected from its secretions by an efficient insulating layer of mucous, enabling most of the small erosions to heal promptly in a few hours. Lack of such a protective mechanism in the duodenal cap

may explain the higher incidence of chronic ulceration in this region.

6. It appears likely, then, that the chain of events beginning with anxiety and conflict and their associated overactivity of the stomach and ending with hemorrhage or perforation is that which is involved in the natural history of peptic ulcer in human beings.

Mittelmann and Wolff[15] conducted a study to ascertain what personality features and emotions predominate in patients with peptic ulcers; what changes in gastroduodenal function are associated with the emotions, and whether such changes may be forerunners of tissue destruction. A series of 30 unselected patients with peptic ulcers, including 27 men and 3 women ranging in age from 19 to 64 years, was studied. Thirteen "normal" subjects with no signs or symptoms referable to the gastrointestinal tract served as a control group. They found that the personality features of these patients were variable and the incidents precipitating the emotional reactions numerous. However, the reactions of intense anxiety, insecurity, resentment, guilt, and frustration were obtained in all. Also common were compensating efforts to bolster self-esteem by a show of independence, self-sufficiency, and perfectionism. The long duration of the personality disturbances demonstrated that the mucosal lesion itself was not responsible for the major emotional conflicts. In all of the patients with peptic lesions it was possible to demonstrate a chronologic parallelism between the onset, recrudescence and course of gastroduodenal symptoms, and the occurrence of untoward emotional reactions. In order to demonstrate that the emotional states described previously were related to the gastroduodenal dysfunction and peptic ulcers in the patients studied, situations were experimentally created which induced destructive emotional reactions and precipitated symptoms when a patient was free of symptoms. Moreover, if such effects, symptoms and tissue defects already existed, all increased in intensity during such experimental procedures. Conversely, in situations which engendered feelings of emotional security and assurance, gastric function was restored toward normal and symptoms eliminated in those patients with symptoms and abnormal function. With a knowledge of the dominant emotions in these patients and some of the circumstances responsible for their being, situations were created in the laboratory which resulted in emotional and gastroduodenal changes like those occurring in the individual's daily experience. Records were made of the behavior and utterances of the subjects and simultaneous records were made of the motility and secretions of the stomach.

The facts revealed by this study again emphasized the occurrence in the same individual of the aforementioned destructive emotions with increased motility and secretion and mucosal circulatory changes in the stomach and duodenum, followed by evidence of gastritis and duodenitis, and ultimately actual ulceration. A comparison of the individual physiologic and emotional changes in normal subjects revealed similar patterns but the changes in the pathologic group were greater in magnitude and duration. The authors, in appraising the data of this study, did not interpret the relation between the emotions as described and functional changes in the stomach and duodenum as one of cause and effect. Instead, both the effects and gastroduodenal changes were viewed as coincident aspects of behavior in reaction to life situations; behavior in some instances compatible with health, and in others resulting in disease.

A large amount of work has been done in order to determine the relation between specific affect and stomach function.

The studies of several investigators on man and on cats and dogs have demonstrated that fears and threats may be associated with reduction in gastric peristaltic activity and decreased secretion with hypoacidity.[14, 16] Many other investigators have described similar changes associated with rage.[17, 18] Bennett and Venables[19] have reported decreased acidity which was later followed by a rise in a subject where disgust was induced by hypnosis. Severe muscular activity as in competitive games may be associated with a hypoacidity.[20]

On the other hand, anxiety in human beings has been shown to be associated with hyperacidity.[21] Similarly, intense hostility may be accompanied by hyperacidity and increased peristaltic activity.[22]

However, Mittelmann and Wolff[15] cite the work of many observers to show that, in many instances, the results of the changes in gastroduodenal functions associated with various affective states are not all in accord. In the study of effects produced by fear, Wittkower[23] concluded that, although fear was always associated with the same effects in the same patient, the reaction in some individuals was associated with hypoacidity, in others with hyperacidity. Mittelmann and Wolff[15] suggest that the apparent discrepancies may be explained by the fact that the reactions to life situations are always complex, often intermingling resentment, fear, feelings of helplessness, vengeance, remorse, and even elation, it is necessary to recognize that some one or two emotions may dominate the reaction. They feel further that it is conceivable that there are individuals who are so constituted that resentment, hostility, anger, and hate never become dominant, so that fear prevails whenever the individual's security is challenged. It is interesting that in Draper's[13] analysis of 80 unselected subjects with peptic ulcer he found that the fear emotion arising from various stimulating mechanisms was the essential point. Since his work is on the largest group of ulcer patients studied with special regard to emotional components, I think it would be highly informative to consider it in some detail especially, as will be later shown, in that it conforms closely with the observations of several other investigators including those of the psychoanalytic school.

First, let us review some of the pertinent facts and theories necessary to the understanding of Draper's conclusions. It is a common observation that instinctively, following the severance of the umbilical cord, the baby seeks the breast in his effort to reestablish promptly his food line. The mother who is actually the only source of nourishment, serves as the object toward which this urgent demand is directed. During the succeeding six or seven years as the variety of other hazards to safety increase, the child still finds in the mother the best guarantee of security against them. In fact, Freud[24] describes the association of the early habits of nutrition with definite gratification of a sexual nature, the so-called "anaclitic" tendency, which is an important part of the infantile sex life. As far as the very young infant is concerned his reliance upon the mother is quite impersonal and it is probable that in his conditioned emotional patterns, the mother represents only a means of survival and not a recognized personality. Eventually, the mother assumes an intensely personal relationship which may even be of a demanding nature so that in the outward relationship to her child, the significance of the mother as a means of survival may disappear entirely. However, in the unconscious levels, the mother remains the symbol of her primary function.[13]

Now psychoanalysis has taught us that substitutes may be adopted for this "mother principle" or "means of survival." In some cases it may consist of surrogates such as a foster mother, grandmother, wife, daughter, sister, nurse or doctor. In other cases symbols such as a social group, business firm, job, college (alma mater), church, food and housing are adopted. Draper[13] feels that when one reads the more intimate histories of ulcer patients, one in confronted by the impression that these individuals, like frightened, neglected children, seem to be continuously striving to recapture and maintain the mother principle.

Alexander,[25] in a study of nine cases (six duodenal ulcers and three gastric neuroses) feels that gastric symptoms often appear in connection with intense oral-receptive tendencies, the wish to be taken care of and loved. He is of the opinion that these are usually repressed to some extent and that in most cases the typical conflict-situation can be described as the rejection of strong-oral receptive tendencies on account of their incompatibility with the aspiration of the ego for independence and activity. He verbalizes the conscious attitude of these patients in the following manner: "I am efficient, active, productive; or I give to everybody, help people, assume responsibilities, like to have people depend on me, like to be the effective leader and self-sufficient, active or even aggressive personality." At the same time, analysis of the unconscious mind reveals exactly the opposite attitude: an extreme and violent craving for love and the need for dependence and help. We now recall the facts observed in the clinic (Cushing, Draper) namely, that these patients characteristically in their actual life relations avoid dependence and assume the exact opposite of the infantile oral-receptive attitude. Instead of receiving, there is in these patients a tendency to give and instead of dependence, the assumption of leadership and responsibility. Alexander feels that these over-compensations must reactively increase in their unconscious the longing for possessive dependence especially since the individuals frequently live beyond their psychic means in the vehement denial of the need for external help. In all of his cases, Alexander found in the unconscious a deep oral regression to the parasitic situation of the infant, which in most cases is incompatible with the attitude and ideals of the adult ego and must of necessity, be rejected. On further analysis, it was shown that the specific reasons for the ego's rejection of these parasitic infantile tendencies involved two predominant natures:

1. A narcissistic injury caused by the infantile claims and manifested on the surface in a sense of inferiority on the one hand, and
2. Guilt and fear on the other.

We come now to what is perhaps the most significant of the various integrations described in this paper. Let us first review what we have so far learned: we have seen that there is a definite relationship between the autonomic nervous system and gastric function and dysfunction. Further, evidence has been cited suggesting the role of the hypothalamus both as the container of the sympathetic and parasympathetic centers and as a seat of certain mechanisms which seem to control various primitive reactions. These reactions are associated in animals with defense or attack and in man with emotional states such as fear and anger. Then, recognizing that the alimentary tract is used by the psychic apparatus with great predilection for the relief of various tensions with strong emotional content, we sought the evidence offered by those workers who inquired into the more precise mechanisms involved. We then reviewed some of the more conclusive experimental works regarding the changes produced in the gastric and duodenal mucosa by the various emotions or affects. Upon examining the conclusions of those investigators who concerned themselves with the personality make-up of the ulcer patients, we find that the conflict situation, in the majority of the carefully analyzed cases, revolves around infantile regressive tendencies rejected by the adult ego. Associated with these tendencies are a sense of inferiority on the one hand and guilt and fear on the other.

Now most clinicians will agree that in a large majority of cases peptic ulcers are due to certain physiologic changes in the secretory and motor functions of the stomach, and possibly also to changes in the blood supply. It is their assumption that the local process, the ulceration of tissue, is the morphologic change which is the final result of certain prolonged dysfunction. Alvarez[26] has suggested that chronic stimulation of the empty stomach is one of the etiologic factors in peptic ulcer. Alexander[25] cites the interesting experiments of Silbermann who produced ulcers in the stomachs of dogs by sham feeding through an artificial esophageal fistula. Palmer[27] and Winkelstein[28] have also offered evidence to support the assumption that one of the causative factors in peptic ulcer formation is due to continuous secretion under the influence of chronic psychological stimuli (oral phantasies and oral tendencies.) Winkelstein observed high acid curves in ulcer patients produced with sham ("psychic") feeding.

At this point we must turn to the psychoanalysts, notably Freud[24] and Abraham,[29] who have demonstrated that the wish to be taken care of and to be helped is emotionally connected in the unconscious with the wish to be fed. That this wish for being fed might serve as a permanent stimulus of the empty stomach and cause its dysfunction is a very interesting speculation and has been suggested by Alexander.[25]

SUMMARY

1. Most important contributions to the literature regarding the etiology and pathogenesis of peptic ulcer are in agreement on one point, that peptic ulcer cannot be explained by the action of any single agent but is the result of the actions and interactions of several factors.

2. An attempt was made to discern what evidence there was to sustain the importance of the psychogenic factor and how psychic influences might mediate changes in the gastric mucosa. We were then able to visualize a series of events wherein emotional conflicts could by stimulation of the autonomic nervous system cause impulses to be conducted along the vagus and sympathetic pathways resulting in dysfunction of the stomach and duodenum.

3. Following the long recognized observations of most clinicians that the peptic ulcer patient is a "high-strung," "nervous" individual, we examined the evidence of workers investigating the more precise psychological makeup of the ulcer patient, and found that the conflict situation in the majority of carefully analyzed, unselected cases, revolved around infantile regressive tendencies rejected by the adult ego.

4. Entering the realm of speculation but with the support of experimental work and certain well established facts of psychoanalysis, we learned that the wish to be taken care of and to be helped (manifestations of infantile regressive tendencies) can be emotionally connected in the unconscious mind with the wish to be fed. Further, that this wish might be the source of permanent stimulation of the empty stomach and result in its dysfunction.

CONCLUSION

It is possible that the major facts involved in the genesis of peptic ulcer are now known. So far, no single theory has been able to withstand the combined critical analysis of the clinician, the pathologist, the physiologist and the neuropsychiatrist. The presence of psychogenic factors associated with this disease process has long been recognized, and it is hoped that enough evidence has been presented in this paper to demonstrate that psychogenic factors may play an important part in the etiology and pathogenesis of this disease. However, we must be aware of the fact that a peptic ulcer is not the organ-symbol of the emotional conflict as is seen for example in certain cases of cardiospasm or globus hystericus. Whereas in conversion hysteria the unconscious tendency may, at times, find direct expression in the physical disturbances, in organic processes controlled by the vegetative nervous system, a longer chain of intermediate physiologic processes may intervene. Thus we may consider an ulcer in the stomach or duodenum as a direct result of a disturbance in the local physiology (motor, secretory, vascular) which disturbances, however, may be precipitated by factors of an emotional or psychogenic nature.

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NEW ORLEANS

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THE ANNUAL MEETING

Many of the members of the State Medical Society look forward to the annual meeting of the organization with the pleasant anticipation of having the opportunity of seeing their old friends and of learning something of the advances in medicine. Unfortunately the number in attendance is not always as large as we would like to see it. As a matter of fact a goal of one hundred per cent attendance might denude the State of Louisiana of capable doctors but we are sure that not one would attend if he had a critically ill patient whom he could not well leave.

The annual meeting this year will be held in New Orleans May 12-14 and while ample hotel space has been promised, nevertheless the exigencies of hotel room shortage make it advisable to make reservations for accommodations at the earliest possible moment.

The scientific program this year has been extensively studied by a committee appointed by President McMahon and will be quite different from the programs as they have been presented in the past. For example, in some of the specialties the program will be entirely devoted to that particular specialty; pediatrics, for instance. In addition to the changes in the program that have just been mentioned, it should be pointed out that a group of some six distinguished and well known out-of-state clinicians have been invited to participate in the presentations. Some of these men will participate in two important symposia that will be given, one on antibiotics and the other on hypertension; both these subjects are of extreme importance at the present time,

The scientific program appears to be outstanding but the social features of the meeting will not be neglected. There will be specialty luncheons and of course the usual president's reception and dinner-dance as well.

It seems that the program of this annual convocation of the State Medical Society, both from a scientific and social aspect, will be outstanding. It certainly will pay the practitioners of medicine throughout the state to attend this meeting. There will be ample opportunity both for culture and for recreation.

BACITRACIN

New and effective antibiotics are being discovered from time to time. Some of these antibiotics are very limited in their field of service, others show remarkable effect on many organisms. One of the latest, apparently very effective of these antibodies was reported on by Johnson, Anker and Meleney[1] in Science this fall. This report aroused a great deal of enthusiasm and there are numerous investigators who are anxious to obtain the material to study its effect in various types of infection. Now Meleney and Johnson[2] report upon the first hundred cases of surgical infections treated locally with this particular medicament. Bacitracin is a filtrate of an aeorbic gram-positive, spore-forming bacillus which has a wide field of antibacterial activity. The preparation has gradually been refined and with this refinement necessarily it has been possible to reduce the size of the dose.

In this present report the drug was used entirely locally, injected directly into furuncles and boils and into deep and superficial abscesses, or applied topically, as in a sty or ulcer of the leg. The cases were not selected and represent the average run of infection which is observed in an ambulatory clinic. Meleney and his associate found that in 31 cases the results of treatment were excellent, in 57 good, in nine questionable and in only three was there no effect. Bacitracin was found effective in most infections which are resistant to penicillin and in some that did not respond to this particular antibiotic and in only a few instances in which bacitracin failed to bring about a cure did the subsequent response to penicillin become effective.

The medicament is given in two forms; an aqueous solution and the other a water soluble ointment. The aqueous solution was injected directly into the lesion, the ointment was used in such sites where occurred ulcers or directly in the eye in the instance of styes and conjunctivitis.

The authors point out that this new antibiotic which is not locally toxic or irritating and is not inhibited by blood or pus, will be of clinical importance if it can succeed where penicillin or the sulfonamides have failed. Their observations, moreover, point out that this preparation is active against many organisms, coccal in nature, which are resistant to the other two important medicaments.

REPORTABLE DISEASE IN LOUISIANA IN 1946

It is rather interesting to note the frequency with which certain diseases have occurred throughout the state in the past year. Excluding the venereal diseases of which syphilis and gonorrhea are diseases which usually lead the list of reportable diseases, it will be found that influenza with 20,108 cases was the most frequent of the diseases that were reported to the State Department of Health. The number of cases of influenza far exceed any other single disease. Of course these figures must be taken with a grain or two of salt as it is quite possible that there were more than 530 cases of mumps in the State of Louisiana in 1946 and undoubtedly more than 3,423 cases of measles or 300 cases of whooping cough, but physicians are wont to forget to report these relatively unimportant childhood disease.

The most surprising feature of the yearly report is the fact that there were 2,078 cases of unclassified pneumonia listed and 613 cases of pneumococcic pneumonia. Without doubt unclassified pneumonia sometimes called atypical or viral pneumonia has become a very real problem. Practically every case of pulmonary tuberculosis is reported and of the respiratory infections there were more of these cases, 2,144, than of the other expressions of respiratory tract diseases.

It is to be noted that there were only

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[1] Johnson, B. A., Anker, H., and Meleney, F. L.: Bacitracin; a new antibiotic produced by a member of the B. subtilis group, Science, 102:376, 1946.

[2] Meleney, F. L., and Johnson, B.: Bacitracin therapy; the first 100 cases of surgical infections treated locally with the antibiotic, J. A. M. A., 133:675, 1947.

206 cases of rheumatic fever listed, 113 of meningitis but most tragic it must be said that there were 381 cases of poliomyelitis in the state, a greater number than ever reported before. There was a great number of persons who suffered from malaria, some 745, but many of these individuals contracted their disease while overseas so that the figures do not actually represent the number of cases of malaria that arise within Louisiana.

Many years ago typhoid fever was the infectious disease most frequently observed; in the past year there were 175 cases and undoubtedly most of these cases could have been prevented had prophylactic measures been employed. On the other hand, a disease which has only within the last two decades achieved prominence, namely typhus fever of which there were 286 cases reported, the preventive measures are not as easily obtained or put into effect as they are with typhoid fever. Tularemia, of which we hear a great deal about at the present time because of the definite satisfactory action of streptomycin, is a relatively infrequent disease with only 43 cases listed in a section of the country in which tularemia is supposed to be rife. Lastly, it might be said of another disease which at one time was frequently observed, namely pellagra, there were only six cases discovered in the state this year and there was not a single case of rabies.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 ANNUAL MEETING GREETINGS FROM THE ORLENS PARISH MEDICAL SOCIETY

The doctors of Orleans Parish look forward with a great deal of pleasure to being host to the members of the Louisiana State Medical Society May 12-14 when the 1947 meeting will be held in this city.

The various committees on arrangements have been very active in making plans for a meeting which will be most interesting, instructive and entertaining.

The limited number of hotel rooms available makes it advisable that all who plan to attend make reservations as soon as possible. This should be done through the office of the secretary-treasurer, however some of the doctors no doubt have connections whereby they can obtain a room through other means and if this is possible and they will do so it would help considerably in securing rooms for all desiring accommodations.

It is hoped that all members of the organization will plan to attend this meeting and they may be assured of a most hearty welcome on the part of the New Orleans doctors.

Your special attention is directed to the following program planned for the 1947 meeting to be held at the Roosevelt Hotel, New Orleans, May 12-14. As you will note, the arrangement for this year is different from that for previous years. It is hoped that we may receive comment from our members concerning this set-up as an effort has been made to have a program which will be of interest and benefit to all concerned. Dr. Donovan C. Browne, Chairman, and other members of the special committee appointed to study rearrangement of the program, are to be commended for their efforts in this regard.

LOUISIANA STATE MEDICAL SOCIETY PROGRAM

1947 ANNUAL MEETING
Roosevelt Hotel

MONDAY, MAY 12:

8:00 a.m.—REGISTRATION—Gold Room.

9:00 a.m.—HOUSE OF DELEGATES—Grand Ball Room.

12:00 noon—LUNCHEON FOR HOUSE OF DELEGATES—University Room.
REGISTRATION—Gold Room.

8:00 p.m.—OPENING MEETING—Grand Ball Room (Joint Meeting with Orleans Parish Medical Society).

1. Call to Order — Rhett McMahon, M. D., Baton Rouge, President.
2. Invocation—Father A. W. Crandell, S. J., New Orleans, Dean, Loyola University of the South.
3. Address of Welcome—Ashton Thomas, M. D., New Orleans, President, Orleans Parish Medical Society.
4. Address of Welcome—Hon. deLesseps S. Morrison, New Orleans, Mayor, City of New Orleans.
5. Response — Gilbert C. Anderson, M. D., New Orleans, President-elect.
6. Drugs Used in Treatment of Heart Diseases—Harry S. Gold, M. D., New York, Associate Professor of Pharmacology, Cornell.
7. Clinico - Pathologic Conference — Chas. E. Dunlap, M. D., New Orleans, Pathologist; Edgar Hull, M. D., New Orleans, Clinician.

TUESDAY, MAY 13:

8:00 a.m.—REGISTRATION—Gold Room.

9:00 a.m.—SYMPOSIUM: ANTIBIOTICS—University Room.

1. Present-day Chemotherapy on Antibiotics—Maxwell Finland, M. D., Boston, Assistant Professor of Medicine, Harvard University.
2. Use of These Agents in Surgery—Champ Lyons, M. D., New Orleans.
3. Use of These Agents in Ear, Nose and Throat—G. F. Joseph, M. D., New Orleans.
4. Use of These Agents in Pediatrics—George Salmon, M. D., Houston, Assistant Professor of Pediatrics, Baylor.
5. Discussion: Questions and answers.

12:00 noon—LUNCHEON FOR GENERAL MEMBERSHIP—Grand Ball Room (Guests of State Society).

1. Epidemiology and Recent Developments in Poliomyelitis—Joseph G. Molner, M. D., Detroit, Associate Professor of Preventive Medicine and Public Health, Wayne University.

2:00 p.m.—SYMPOSIUM: HYPERTENSION—University Room.

1. Application of Fundamental Knowledge of Hypertension and Its Treatment—Raymond L. Gregory, M. D., Galveston, Professor of Pharmacology, University of Texas.
2. Surgical Treatment of Hypertension—L. J. O'Neil, M. D., New Orleans.
3. The Use of Drugs in the Treatment of Hypertension—M. E. St. Martin, M. D., New Orleans.

Intermission — View Technical Exhibits.

4. Liver Disease Illustrated by Cases —Presented by Residents and discussed by E. S. Kagy, M. D., J. R. Snavely, M. D., C. A. Jones, M. D., Ambrose Storck, M. D., Roy H. Turner, M. D., New Orleans.
Discussion: Questions and answers.

8:00 p.m.—PRESIDENT'S RECEPTION AND DINNER DANCE—Grand Ball Room (Members and guests of the Society)

WEDNESDAY, MAY 14:

8:00 a.m.—REGISTRATION—Gold Room.

9:00 a.m.—HOUSE OF DELEGATES—Grand Ball Room.

SCIENTIFIC PROGRAM:

I. PAN-AMERICAN ROOM.

1. Intestinal Obstruction—M. L. Michel, Jr., M. D., New Orleans.
2. Critical Analysis of 300 Cases of Hip Nailing—I. L. George, M. D., Baton Rouge.

Intermission — View Technical Exhibits.

3. Extraintestinal Amebic Infection—W. R. Akenhead, M. D., New Orleans.
4. Drug Reactions and Their Treatment with Special Reference to the Use of BAL in Heavy Metal Poisoning — Barrett Kennedy, M. D., and V. Medd Henington, M. D., New Orleans.

II. ROOM H.

1. Radiation Treatment of Cancer —S. F. Hatchette, M. D., Lake Charles.
2. Roentgenological Aspects of Pneumonias—Leon J. Menville, M. D., New Orleans.
3. Hypertrophic Pyloric Stenosis—H. O. Barker, M. D., Alexandria.
Discussion—H. H. Hardy, M. D., Alexandria.
Followed by — H. A. White, M. D., Alexandria.

Intermission — View Technical Exhibits.

4. Hematuria — C. A. Frederick, M. D., Lake Charles.
5. Management of Prostatism—U. S. Hargrove, M. D., Baton Rouge.

12:00 noon—LUNCHEONS FOR MEMBERS OF SPECIAL SOCIETIES.

WEDNESDAY, MAY 14:

2:00 p.m.—SCIENTIFIC PROGRAM:

I. PAN-AMERICAN ROOM.

1. The Present Status of the Poliomyelitis Problem—V. Lippard, M. D., New Orleans.
2. Amebiasis in Children—Maud Loeber, M. D., and Joseph S. D'Antoni, M. D., New Orleans.
3. Marrow Infusions and Transfusions—Max Sutton, M. D., and George Kelley, M. D., New Orleans.
4. Air-Block in the Newborn Period—George Salmon, M. D., Houston, Assistant Professor Pediatrics, Baylor.
5. Business Meeting of Louisiana Pediatric Society.

II. GRAND BALL ROOM.

1. Irradiation Therapy of Malignant Neoplasms of the Female Genitalia—M. Garcia, M. D., and J. V. Schlosser, M. D., New Orleans.
2. Pruritus Vulvae and Ani—M. T. Van Studdiford, M. D., and L. D. McLean, M. D., New Orleans.
3. Circumvallate Placenta; Symptoms and Management—Robert D. Mussey, M. D., Rochester, Minn., Professor of Obstetrics, University of Minnesota Graduate School of Medicine.
4. The Rh Factor in Clinical Obstetrics and Gynecology—J. W. Davenport, Jr., M. D., New Orleans.
5. Motion Picture: Ligation of the Vena Cava—Conrad G. Collins, M. D., New Orleans.

III. ROOM H.

1. Congenital Ocular Anomalies Resulting from (a) Premature Birth, (b) Rubella Infection of Mother During Pregnancy—W. R. Buffington, M. D., New Orleans.
   Discussion—George L. Hardin, M. D., New Orleans.
2. Functional Conditions of the Nose—Joseph Stamm, M. D., New Orleans.
3. Hoarseness—George J. Taquino, M. D., New Orleans.

Intermission — View Technical Exhibits.

4. Treatment of Anemia—Norton W. Voorhies, M. D., New Orleans.
5. The Present-day Treatment of Malaria — A. J. Walker, M. D., New Orleans.

WEDNESDAY, MAY 14:

6:30 p.m.—DINNER FOR DIPLOMATES OF AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY—Antoine's Restaurant.

COCKTAIL PARTY FOR MEMBERS OF LOUISIANA PEDIATRIC SOCIETY—Metairie Country Club.

8:00 p.m.—DINNER FOR MEMBERS OF LOUISIANA PEDIATRIC SOCIETY—Metairie Country Club.

1. Autopsy Findings in Premature Infants—James Arey, M. D., New Orleans.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

April 11,	Special Meeting, Orleans Parish Medical Society with the Louisiana Physicians Service, Incorporated, Hutchinson Memorial Auditorium, 8 p. m.
April 14,	Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
April 15,	I. C. R. R. Hospital Staff, 12:30 p. m. Charity Hospital Medical Staff, 8 p. m.
April 16,	The Orleans Society of X-Ray Technicians, 7:30 p. m. Charity Hospital Surgical Staff, 8 p. m.
April 17,	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
April 18,	Lakeshore Hospital Staff, 8 p. m. Executive Committee, Hotel Dieu, 8 p. m.
April 21,	Hotel Dieu Staff, 8 p. m.
April 22,	Baptist Hospital Staff, 8 p. m.
April 23,	French Hospital Staff, 8 p. m. Catholic Physicians' Guild, 8 p. m.
April 24,	DePaul Sanitarium Staff, 8 p. m.
April 25,	L. S. U. Faculty Club, 8 p. m.

New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
May 1, Clinico-pathologic Conference, Touro Infirmary, 12 noon.
May 2, Ochsner Clinic Staff, 8 p. m.
May 5, Board of Directors, Orleans Parish Medical Society, 8 p .m.
May 6, Orleans Parish Radiological Society, 7:30 p. m.
Eye, Ear, Nose and Throat Staff, 8 p. m.
May 7, Mercy Hospital Staff, 8 p. m.
EVERY THURSDAY—Medical Toastmasters, 8 p. m.

NEWS ITEMS

The Medical Toastmasters have discontinued meeting at the New Orleans Athletic Club at 6:30 p. m. They are now meeting in the Hutchinson Memorial Auditorium at 8 p. m.

Dr. Howard Mahorner was host to the members of the Surgeons Travel Club, February 11-14.

Dr. B. Bernard Weinstein attended the meeting of the Western Branch of the American Society for the Study of Sterility at Los Angeles. February 7-8. Dr. Weinstein prosented a paper on "The Surgical Management of the Tubal Factor in Serility."

The annual meeting of the New Orleans Society of Neurology and Psychiatry was held on January 23. The following new officers were elected: Dr. Henry O. Colomb, president; Dr. Sam Nelken, secretary-treasurer; Dr. Dean Echols, the retiring president, to the executive committee, which already included Dr. C. Grenes Cole and Dr. Edmund Connely.

Dr. William H. Block has been honored with a life membership in the Alumni Association of the University of Maryland. Dr. Block has been an active member of the Association for fifty years.

CHANGES IN MEMBERSHIP ROLLS

Three associate members, Drs. Brent Fox, Charles O'D. Lilly and Wm. H. Newman, were transferred to intern membership.

Two physicians, Drs. Roscoe Pullen and Robert M. Rose, were reinstated to active membership.

Two active members, Drs. John T. Crebbin and M. M. Odom, were elected to inactive membership.

One associate member, Dr. Patrick Riley, and two intern members, Drs. Ben F. Thompson and David F. Watson, were removed because of removal from New Orleans.

CHANGES OF ADDRESS

Dr. Robert M. Kimball—1430 Tulane Avenue.
Dr. Malcolm P. Schwarzenbach—244 Oliver Street, Algiers.

NOTICE

The Library is in need of November 1946 issues of the New Orleans Medical and Surgical Journal for exchange purposes. If you have a copy available, which you do not need, we would appreciate your forwarding it to the Library. Thanks.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

PRESIDENT RHETT McMAHON

Dr. Rhett G. McMahon, the President of the Louisiana State Medical Society, was born on 22 July, 1891 in Vinton, Louisiana. His preparatory work was done at the Mt. Talmalpais Military Academy at San Rafael, California. His academic work was completed at the University of California and Louisiana State University. He was graduated from Tulane Medical College in the Class of 1921, and afterwards interned at Charity Hospital for two years.

He is a member of the East Baton Rouge Parish

Medical Society (Past President), the Sixth District Medical Society, The Louisiana State Medical Society (President), the Southern Medical Society, the American Medical Association, the American College of Surgeons (Fellow), the Central Association of Obstetricians and Gynecologists and the Louisiana Obstetrical and Gynecological society.

For a number of years he was a Councilor of the State Medical Society, is now vice-president of the Louisiana State Board of Medical Examiners, president of the Hospital Service of Baton Rouge and a member of the Elks Club and Baton Rouge Country Club.

During the war years he served with honor and distinction as a member of the Selective Service Board and as a member of the Procurement and Assignment Board.

Dr. McMahon has always been a leader in civic affairs and is a large contributor to the upbuilding of his native city.

Since his graduation Dr. McMahon has had a number of post graduate courses, particularly in the Post Graduate School of Medicine and Woman's Hospital in New York and also the Lying-In Hospital in Chicago and Barnes Maternity Hospital in St. Louis.

Dr. McMahon has been well trained for the position that he occupies by having served two years as president elect of the Louisiana State Medical Society. The Society is most fortunate in having elevated to the presidency a man of such excellent traits of character, indefatigable energy and executive ability.

RAPIDES PARISH MEDICAL SOCIETY

The regular monthly meeting of the Rapides Medical Society was held Monday, March 3, 1947 at 7:30 p. m. at the Baptist Hospital in Alexandria, Louisiana.

A scientific and business program was presented.

THE FOUNDATION HOSPITAL

A staff meeting of this hospital was held on Friday, March 7. The program consisted of the following presentations: Personality Disorders in Gastroenterology by Dr. Albert J. Sullivan; the Rh Factor, by Dr. Julius Davenport; Foreign Bodies in the Food and Air Passages by Dr. Clarence H. Steele.

TOURO INFIRMARY

A regular meeting of the Medical Staff of Touro Infirmary was held on Wednesday, March 12. The Department of Pathology presented a clinico-pathologic conference with a clinical discussion by Dr. John Dyer. Dr. Mims Gage gave a case report on acute pancreatitis. Drs. J. D. Rives and Allan M. Goldman presented a case of mesenteric thrombosis with survival.

NEW SUPERINTENDENT OF BAPTIST HOSPITAL

Reverend Frank Tripp, of Montgomery, has accepted the post of superintendent of the Baptist Hospital of New Orleans and will assume his duties May 1. Dr. L. J. Bristow will remain with the institution in an executive capacity.

MATAS MEDAL

Dr. Emile Bloch, chairman of the committee which has to do with the award of the Matas Medal, announced that this honor will be given to Dr. Robert E. Gross, of Boston, Mass., whose work on the operative treatment of congenital heart disease has been outstanding.

THE CIRCLE OF THE L. S. U. MEDICAL SCHOOL

Doctor William Dock, Professor of Medicine, Long Island College of Medicine, Brooklyn, N. Y., delivered the annual lecture of The Circle, honorary medical society of the Louisiana State University School of Medicine, in the Auditorium of Charity Hospital. The subject of his lecture was proteinuria and edema.

Doctor Dock is well known for his investigations in the field of internal medicine and has recently written extensively on the ambulatory treatment of heart disease and other conditions which had traditionally required long periods of rest in bed. Before becoming associated with the Long Island College of Medicine, he was a member of the medical faculties at the University of California and Cornell University. While in New Orleans, he also took part in the Graduate Medical Assembly.

The following students, selected on the basis of outstanding scholarship, are members of The Circle:

Richard L. Bagnetto, New Orleans, La.
Lawrence G. Bole, New Orleans, La.
Oscar Wharton Brown, Baton Rouge, La.
John W. Fisher, Dallas, Texas.
Nicholas J. Olivier, New Orleans, La.
Louis G. Pascal, New Orleans, La.
Eliza Ann Sarton, Alto, La.
Chenet J. Schexnaildre, Metairie, La.
Robert R. Sills, New Orleans, La.
Irving Singer, New Orleans, La.
Hannah B. Moody, New Orleans, La.

SOUTHERN SOCIETY FOR CLINICAL RESEARCH

The first annual meeting of the Southern Society for Clinical Research was held recently in New Orleans. The purpose of the society is encouraging research in the various medical sciences. The following officers were elected:

President—Dr. Tinsley R. Harrison, Dallas, Texas.
Vice-President—Dr. William J. Darby, Jr., Nashville, Tenn.
Secretary and Treasurer—Dr. Thomas Findley, New Orleans, La.
Councilors—Dr. Paul B. Beeson, Atlanta, Ga.; Dr. Robert M. Moore, Galveston, Texas; Dr. Joseph W. Beard, Durham, N. C.; Dr. Harold B. Greene, Winston-Salem, N. C.

The Founders' Group consists of approximately forty men from the various medical schools in the South but membership is not limited to academic institutions.

POSTGRADUATE REVIEW IN NEOPLASTIC DISEASES

There will be held on April 7-12 a review in neoplastic diseases given by the Tulane University of Louisiana, Division of Graduate Medicine, and sponsored by the Orleans Parish Unit and the Louisiana State Division of the American Cancer Society. Dr. Ambrose H. Storck is in charge of this program and he, together with members of the program committee, has provided a very excellent series of talks, lectures and demonstrations by various physicians, some of whom are of outstanding prominence in their particular field. Dr. Fred W. Rankin, who was a Brigadier General in the war in charge of the surgical division of the Surgeon General's Office, is an example.

POSTGRADUATE COURSE IN DISEASES OF NUTRITION AND METABOLISM

Under the chairmanship of Dr. Grace A. Goldsmith there was held at the Tulane University of Louisiana, Division of Graduate Medicine, a most excellent course in metabolic and nutritional diseases. Nutritional metabolic problems of the child were discussed as well as those that occur in adults.

INSTRUCTIONAL COURSE IN ALLERGY

The Mississippi Valley Sectional Instructional Course in Allergy will be given under the auspices of the University of Kansas School of Medicine, on May 5-8 inclusive, at the University of Kansas Hospitals, in Kansas City, Kansas, and is sponsored by the American College of Allergists.

A well balanced program has been planned specifically to acquaint the physician with the basic principles of diagnosis and management of allergic diseases.

Round-table discussions, laboratory, and clinical sessions will be held, in addition to formal lectures covering the practical application of the various phases of this subject. Whether you are in the practice of general medicine or limiting your practice to pediatrics, otolaryngology, or dermatology, you will be interested in this course. It was arranged in direct response to various requests from physicians in the above named specialties.

A more detailed program may be had by writing the Chairman. The fee for the course is $50.00 payable at the registration desk at the University of Kansas School of Medicine, Kansas City, Kansas. Applications for the course and for hotel reservations should be placed with the Chairman of the Program Committee, Orval R. Withers, M. D., 1418 Bryant Building, Kansas City 6, Missouri.

MEDICAL DEPARTMENT OF U.S.A.

Highlights of the benefits to The Medical Department of the proposed legislation now before Congress (HR 2536) on procurement, promotion and elimination of Regular Army officers are outlined in the following statement recently (14 March 47) issued by the Office of The Surgeon General:

The bill introduced in Congress to provide for the procurement, promotion and elimination of Regular Army officers and for other purposes provides that Section 10 of the National Defense Act be amended to read: "The Medical Department shall consist of Surgeon General with the rank of major general, four assistants with the rank of brigadier general, one of whom shall be an officer of the Dental Corps" is amended to read: "The Medical Department shall consist of one assistant with the rank of major general who shall be an officer assigned to the Dental Corps, and three assistants with the rank of major general or brigadier general as determined by the Secretary of War."

In addition it authorizes a strength of active permanent general officers of the Regular Army in the Medical Corps equal to 3/4 of 1% of the authorized active commissioned strength of the Medical Corps of the Regular Army. Of a total authorized strength of active general officers there is authorized 50% in the grade of major general and 50% in the grade of brigadier general.

CIVIL SERVICE EXAMINATION FOR MEDICAL OFFICER POSITIONS

The U. S. Civil Service Commission has announced an examination for filling Medical Officer positions.

These positions are located in various Federal agencies, in Washington, D. C., in the U. S. Public Health Service and the Indian Service, throughout the United States; and in the Panama Canal Service, in the Panama Canal Zone. Salaries range from $4,149 to $5,905 a year, with higher salary rates for the Canal Zone positions. No written test will be given for these positions. To qualify, all applicants must be graduates of a medical school of recognized standing and must also meet other requirements which include experience and training in the field of medicine. The age limits are 45 years for Panama Canal Zone positions and 62 years for other positions. These age limits are waived for persons entitled to veteran preference (up to the age of 62 for the Panama Canal Service and without limitation for other agencies).

Interested persons may secure information and application forms from most first- and second-class post offices, from Civil Service regional offices, or from the U. S. Civil Service Commission, Washington 25, D. C. Applications must be filed not later than April 22, 1947.

C. JEFF MILLER MEMORIAL LECTURE

There was delivered by Dr. Emil Novak of Johns Hopkins School of Medicine the C. Jeff Miller Memorial Lecture Friday, March 21. Dr. Novak spoke on the subject of "Carcinoma of the Uterus". Following the formal lecture there was presented to the Tulane Medical School, by Elizabeth Miller Robin, a very handsome portrait of her father, the late Dr. Miller, Professor of Gynecology at Tulane at the time of his death.

ANNUAL MEETING, AMERICAN COLLEGE OF CHEST PHYSICIANS

The Thirteenth Annual Meeting of the American College of Chest Physicians is scheduled to be held at the Ambassador Hotel, Atlantic City, New Jersey, June 5-8. An interesting scientific program has been planned for this meeting. Prominent speakers from other countries will present papers.

The oral and written examinations for Fellowship will be held on the first day of the meeting, June 5. Applicants for Fellowship in the College who plan to take these examinations should communicate *at once* with the Executive Secretary, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

NEWS ITEMS

Harofe Haivri (The Hebrew Medical Journal) which is dedicated to the continued growth of Hebrew medical literature, has concluded its nineteenth year of successful publication, under the editorship of Moses Einhorn, M. D. of New York.

Mr. Francis C. Brown, president of Schering Corporation of Bloomfield and Union, New Jersey, announces an additional number of grants for endocrine and pharmacological research studies awarded by Schering to research foundations, medical and pharmacy colleges.

"The Clinical Use of Androgens in the Female" has been selected as the subject for the 1947 Schering Award Competition among medical students. Cash prizes of $500.00, $300.00 and $200.00 will be given to the prize winners.

The annual meeting of the American Hygiene Association will be held at the Hotel Statler, Buffalo, New York. April 27-May 3. At the same time there will be held other meetings, such as the American Association of Industrial Dentists and a conference of Governmental Industrial Hygienists.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending February 8 there were 147 deaths in the City of New Orleans as contrasted with 109 the previous week. Of these deaths 101 were in the white population and 46 in the colored with 17 of these being children under one year of age. For the succeeding week, February 15, the death rate was approximately the same as for the previous week, 149 deaths being reported this week, two less in the white and four more in the colored population, with two less infant deaths. The week of February 22 the death rate in the city was almost static as for the third week approximately the same number of people expired. In this particular week of the 144 deaths, 94 were in the white population and 50 in the negro with 18 of the deaths being in children under one year of age. The March 1 report showed very little change. The total number of deaths was up to 155, most of the increase being in the white population, 110 of whom succumbed this week with 45 negro deaths and of the total 13 were in children under one year of age. The number of deaths for this week corresponded exactly with the three year median for this particular week. Again there was very little change in the total number of deaths the week ending March 8. There were 152 people who died in New Orleans this week, divided 92 white, 60 non-white and 26 of whom were children under one year of age.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending January 25 showed only three diseases reported in numbers greater than ten, namely, 32 cases of pulmonary tuberculosis, 29 of influenza and 14 of chickenpox. For the week ending February 1 there was a marked increase in the number of reportable diseases. Cancer lead the list with 88 cases, followed by 82 of pulmonary tuberculosis, 58 of unclassified pneumonia, 41 of measles, 33 of chickenpox, 29 of influenza, 25 of pneumococcic pneumonia, 19 of scarlet fever, 14 of diphtheria, 13 of whooping cough and 12 of typhus fever. Nine of the typhus fever cases came from Orleans Parish. The following week, February 8, amebiasis with 23 cases reported, was the only disease listed in the correct weekly morbidity report, which exceeded ten in number. The weekly morbidity report for the week closing February 15 again listed only a very few diseases exceeding ten in number. These were measles with 23 instances, pulmonary tuberculosis with 14 and hookworm infestation with 11. One case of poliomyelitis was reported this week from St. Landry Parish. For the week which terminated February 22 cancer lead the reportable diseases with 57 cases followed by 56 of pulmonary tuberculosis, 28 of influenza, 25 of unclassified pneumonia, 13 of pneumococcic pneumonia and ten of diphtheria. The last report available, which is for the week which closed March 1, pulmonary tuberculosis was first with 56 cases, followed by influenza with 54, cancer with 48, chickenpox with 46, measles with 27 and unclassified pneumonia with the same number, followed by whooping cough with 19, septic sore throat with 16, scarlet fever with 12 and diphtheria with ten. Of the rarer diseases there were five cases of meningococcic meningitis, four of undulant fever and seven of typhoid fever.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending January 31, 1947.

	Total This Month	Total Previous Months	Total To Date 1947
CHANCROID	58	-	58
GONORRHEA	1284	-	1284
GRANULOMA INGUINALE	9	-	9
LYMPHOPATHIA VENEREUM	9	-	9
SYPHILIS	966	-	966

NATIONAL MEETINGS

Following are reports of recent meetings held in Chicago. These conferences were attended by several of our members and we are indebted to Dr J. P. Sanders, member of the Committee on Rural Health, for preparation of these data.

NATIONAL CONFERENCE ON RURAL HEALTH SECOND ANNUAL MEETING HELD IN CHICAGO FEBRUARY 7, 8, 1947

Dr. F. S. Crockett, Chairman, opened the meeting at 9:45 a. m. and outlined some of the needs on rural health and rural medical service. Among things he stated that there should be a creation of a department of rural medical service that would study rural needs which include health, medical, social, economic and other problems of the community and not those of medicine alone. He stated that these were so entwined that it is impossible to segregate one from the other.

The welcome address was given by Dr. George F. Lull, Secretary and General Manager, American Medical Association. He welcomed the organization in behalf of the American Medical Association, said that the American Medical Association is extremely interested in all medical efforts particularly in the medical service council and the work of our organization.

Dr. Lull's talk was followed by a talk by Mr. Albert S. Goss on the farmer and the medical service program. Mr. Goss is master of the National Grange from Washington, D. C. and he brought out the following points:

(1) That the farmer's ability to pay before the war was $200.00 per capita as compared with $800.00 per capita with the rest of the population. That in 1946 with increased farm prices his income was only $740.00 per capita as compared with $1,260.00 per capita.

(2) That the farmers are usually too far from medical centers to receive good medical care. That on the average there is one doctor to 1,700 farm people whereas the urban areas have one doctor to 650 people. That there is only one bed per thousand people in rural areas whereas good medical care requires that there be three or four beds per thousand.

(3) Doctors do not like inconveniences and low income.

(4) Farm accidents are 53 per thousand in comparison to urban centers that have only 17 accidents per thousand though the farmers

accidents are usually more minor than those of the city brother.

(5) Farmers are not as healthy as city folks. In the Armed Services 41 out of 100 were rejected by induction boards while only 25 per 100 were rejected from the cities.

(6) The cost of adequate medical care has increased considerably.

(7) People are turning to cooperative medicine.

Finally Mr. Goss suggested that all the farm groups including the farmers union, the schools, the churches and the medical societies be organized to solve the problem and avoid State control.

Methods of bringing and holding doctors in rural areas was discussed by Dr. Fred A. Humphrey from Fort Collins, Colorado. He brought out the following points:

(1) How are we going to keep them on the farm?

(2) What is a rural community?

(3) Two groups are interested—the farmers themselves and the doctors involved.

(4) That the general plans should be flexible enough to satisfy the needs of any rural community.

(5) The rural people themselves must feel the responsibility for proper medical care.

(6) Residencies should be given in general hospitals on general practice.

(7) The prestige of the general practitioner must be increased.

(8) That the general practitioner must be given extra training probably for short periods.

Dr. Humphrey's discussion was followed by a talk by Mr. Chester of the Missouri Farm Bureau Federation, Jefferson City, who brought out the following interesting facts:

(1) There is a constant drainage of doctors from rural areas.

(2) Osteopaths are practicing medicine in many rural areas.

(3) There is one doctor to every 1,500 people in the rural areas whereas there is one doctor to 430 people in the most populous rural area and that 43 per cent of the doctors are over sixty year of age.

(4) That doctors are human beings like anybody else and want good income, good roads, good equipment, hospital facilities and prepayment medical plans.

(5) That subsidies should come only as a last resort.

(6) That every effort be made to increase the farmer's income.

(7) That better roads be provided.

(8) Better living conditions are needed for the doctor and his family as well as the community as a whole.

He made the following recommendations:

(1) That young doctors be interested in rural medical education and not the specialities while in school.

(2) That medical education stress more rural practice.

(3) That rural internships be provided.

(4) That doctors be picked out for leadership.

(5) That local residents quit by-passing their local doctors for the more urban centers.

(6) That prepayment medical plans be put in operation.

Hospital Facilities and Health Centers for Rural Areas were discussed by Mrs. Roy C. Weagly, President, The Associated Women of the American Farm Bureau Federation, Hagerstown, Maryland.

Mrs. Weagly brought out the following points:

(1) That the Hospital Construction Act, House Bill No. 725, commonly known as the Hill-Burton Act, will help build non-profit hospitals all over the country.

(2) That the rural areas should get their share equal with urban areas.

(3) That the general over-all plan and study should be made first—before construction is started.

(4) That all sorts of hospitals should be taken care of such as general hospitals, tubercular hospitals and others that may be necessary. That general care for patients such as emergency and minor cases should be treated in these hospitals.

Mr. Fichter was represented by Mr. Halverson from his own office, representative of the Ohio State Grange. He stated:

(1) That a state survey of Ohio is being made in order to find where hospitals are actually needed.

(2) That state aid and local capital are being provided.

(3) That farm representatives should be on all of these hospital boards.

Dr. Robin D. Buerki, Dean, Graduate School of Medicine, University of Pennsylvania, Philadelphia, Member Advisory Board, Federal Hospital Council, brought out the following points:

(1) That the Federal Hospital Council will not support any type of poor medical care.

(2) That before any hospital is built a plan must be developed whereby the maintenance of the hospital shall be cared for which ordinarily amounts to one-third of the original cost per year.

(3) That too many specialists are being trained and not enough doctors.

(4) That the specialist be limited entirely to his specialty.

(5) That there must be a change in the viewpoint relative to rural medical service.

(6) That the United States now has the best medical service in the world.

VOLUNTARY MEDICAL PREPAYMENT PLANS AS THEY APPLY TO RURAL COMMUNITIES

Mrs. Gladys T. Edwards, Director of Education, Farmers Educational and Cooperative Union of America, Denver, Colorado, was not present but was represented by Mr. Birks of her office.

(1) That it is now time for action instead of talking.

(2) That the economic condition of the rural people should be raised and that the biggest cause for the lack of proper medical care is due to lack of income. He stated that one-half of the farm people of the United States have an income of less than $1,000.00 per year per family and that some have less than $400.00 per year.

(3) That farmers have less income than their city brethren.

(4) That the farmers' union and other farm organizations are in favor of a voluntary prepayment medical plan.

(5) That we must increase the farmers' income if we are to bring up to the proper standing.

(6) That they will pay health services cooperatively because the cost is smaller.

Dr. James R. McVay, Vice Chairman, Council on Medical Service, American Medical Association, Kansas City, Missouri, stated that:

(1) Hospital and prepayment medical plans have been working—the first originated in 1882 by North Pacific Railroad. It was followed by the Blue Cross plan in 1939 and state plans are being carried out in Washington and Oregon from about the same year.

(2) That the veterans are turned back to the home town doctor.

(3) That what we need is cooperation, study and expansion.

(4) Nursing and dental care as well as medical care is being tried out in some states while others only cover surgical, obstetrical and other catastrophic illness.

(5) Premiums run from $1.25 per person to $4.85 and $3.00 per family to $10.40 per family per month depending upon the type and amount of services rendered.

(6) That the members of the medical profession must be kept informed as well as the prepayment consumed.

Mr. J. S. Jones, Secretary, Minnesota Farm Bureau Federation, St. Paul, brought out that this meeting is a healthy indication that the Blue Cross and other hospital plans have been in operation for many years and should be combined with prepayment medical plans.

(2) That large groups should work cooperatively.

(3) That no one wants Government in Medicine.

REPORT ON ROUND TABLE DISCUSSION

(1) That the Hill-Burton Bill provides for community clinics, hospitals, hospital-clinics and public health centers.

(2) There is $75,000,000 appropriated yearly for five years for hospital construction.

(3) That an equal amount of money must be raised by the State and by the community.

(4) That a complete over-all survey must be made before construction starts and this must be done by the State Hospital Council.

(5) That interested farm groups must be contacted and gotten interested.

(6) The following four-point program was recommended:

  (a) That the Hill-Burton plan should be followed.

  (b) Educate the population as to what is being attempted and wanted.

  (c) That the community itself must take action.

  (d) That there must be cooperation with State groups.

In the discussion Dr. Voss brought out that geriatrics—medical care and otherwise had been taken care of and that there was no provision made for convalescent care.

Dr. Shaw, of Iowa, pointed out that the bill provided for fifty or more beds when many times in rural areas a hospital of fewer beds is actually needed and where a hospital with only twenty-five or thirty beds would be capable of doing fine work but the cost of beds can not be more than in larger hospitals.

Dr. Walpeck of Salt Lake City, Utah, stated that mining towns and communities have to be cared for. That it is often more practical to send the specialist to the small hospital than to move the patient to a larger medical center.

Dr. Davis of the Federal Hospital Council said that there is no limit on number of beds. It depends entirely upon the needs of the community and that he is against any plan that requires a community to build a hospital larger than it actually needs.

Methods of bringing and holding physicians and dentists in rural areas by Mr. J. S. Jones and Dr. H. B. Mulholland.

They recommended the following:

(1) The development of State and County Health Councils.

(2) The development of health facilities in rural areas with stress on good income for farm groups.

(3) That a well planned program be developed.

(4) That the medical schools teach more of the economic side of medicine.

Mr. Lloyd Jones brought out that these facts should be carried back home to our people, the doctors and others.

Mrs. Goss stated that we must develop the National, State and County Councils with all groups interested brought in—the medics, farmers, farmers' unions, P.T.A.

Professor Mather of Pennsylvania stressed the point that the loyalty of the farmers is not to the county but to the entire community and that community pride should be stressed.

Dr. Miller of Kentucky also discussed community loyalty and also brought out the point that the living place for the doctor and his family is important and that the community must be made aware of this condition.

Mr. Halverson of Ohio suggested the point that scholarships be awarded the rural students who would come back and practice in the rural areas.

Dr. Panquest of Mississippi stated that this state is awarding such scholarships of $5,000.00 per student with an agreement that he return and practice in a rural area from two to five years.

VOLUNTARY PREPAYMENT MEDICAL PLANS

Dr. James F Doughty of Tracy, California, read a report including the following:

(1) That all prepayment plans be extended on a statewide basis.
(2) That prepayment medical plans be put on an educational basis.
(3) Plans be sound financially and as liberal as possible.
(4) To be complete and adequate.
(5) To take care of the general practice particularly in rural communities.
(6) That the community study its problems and develop its own plans.
(7) That it be voluntary and on a non-profit basis.

Dr. Davis brought out that the plans should be approved by the County and State Medical Societies.

Dr. Scott of New Jersey defended the law in his state which provides that these participating insurance companies should have a majority of medical men on their governing boards.

NURSING NEEDS OF RURAL COMMUNITIES

Mrs. Charles W. Sewell read the following report:

(1) Education for nurses must start in the home.
(2) That high school nursing and home nursing should be started in high schools.
(3) Programs should be developed for nursing in rural areas.
(4) Practical nurses or nurses' aides should be trained.
(5) Commensurate pay for training and services rendered should be encouraged.

Dr. Glem Davis of Michigan brought out the difference in trained nurses which require three years' full time training and a nurses' aide which requires one year, part time.

Dr. Minch of Michigan stated again that the P.T.A.'s, schools and teaching institutions should be interested in nursing care.

Dr. Bernard of Minnesota stated that nurses should be trained in rural hospitals.

HEALTH COUNCIL AS AGENCY FOR PROMOTING RURAL HEALTH

Dr. J. Paul Jones from Alabama read a report in which was recommended the following:

(1) Organization of the health communities.
    (a) By those who give and those who receive medical care.
    (b) Governmental agencies to assist in this work.

Objectives

(1) Interchanging of opinions.
(2) Medical care curricular.

Functions of the Health Council:

(1) Make surveys and find the needs.
(2) Obtain needed facilities and personnel.
(3) Plan ways and means of raising money and operation.
(4) Dissemination of information and promote meetings.

Miss Becker of Nebraska stressed the importance of dissemination of information. She stated that 5,000 county agencies in the United States could be used as well as the P.T.A. organization in getting this type of information across.

Dr. Henry of Little Rock, Arkansas, stated that 80 per cent of his state was rural and that their organization was already working together on a cooperative basis.

Mr. Goss said that the councils should definitely be on a county level.

A number of others spoke in favor of these health councils for rural areas and Father Butler, of Tennessee, stressed the importance of the use of the churches in the rural communities for the dissemination of information particularly in the rural churches.

MEDICAL CARE FOR LOWER INCOME GROUPS

Mr. Owens and Mr. Anderson stated that there is too little time and the definitions are too vague for as complete a study as is necessary to make definite recommendations. For example, they stated that they did not know what constituted medical care, whether it was complete, whether it included hospitalization, dental and nursing care or whether it should be limited to certain types of medical care alone. Then they brought up the question of what is a low income group.

The following suggestions were offered:

(1) That there be a sliding scale of fees.
(2) Prepayment medical insurance.
(3) Tax support for indigents.
(4) Adequate medical care for all.

The following recommendations were made:

(1) That health is a result of economic status.
(2) That low income groups probably cover 85 per cent of all population.

(3) That a comprehensive study be made of the medical care needs of each community.
(4) That complete health care be planned for all.
(5) That we either have a voluntary prepayment medical insurance supplemented by tax funds or adopt a national health plan by taxation.

Dr. Garnett, of Virginia, stated that a great many people in his state are not getting adequate medical care, that prepayment medical insurance is definitely important and that one-third more children are in the poorer income group than in the higher brackets. He stated that in his state there are 100,000 white families and 65,000 negro families in the lower income group.

Reverend Adams, or St. Louis, represented the Catholic conference. He thanked the A. M. A. for its interest and activities. He thought that there was not time for complete reports but that a study should be continued. He stated that one-third of the cities are slum districts, that we should have more preventives in schools and definite health programs. He stated that we should definitely foster local groups.

At a breakfast in the Palmer House on February 8, a regional meeting was held of the representatives from Florida, Georgia, Alabama, Mississippi, Louisiana and Arkansas with the following representatives present.

Mr. J. Paul Jones, Camden, Alabama, Chairman
Dr. Henry, Little Rock, Arkansas
Dr. Reed, Arkadelphia, Arkansas
Dr. Holland, Leesburg, Florida
Dr. Guy Jones, Lockport, Louisiana.
Dr. M. C. Wiginton, Hammond, Louisiana
Dr. J. P. Sanders, Shreveport, Louisiana

It was decided that a regional meeting be held in the near future probably in Biloxi, Mississippi.

Regional reports were made from most of the states of the United States.

Reports were given upon what is going on in the working out of the Hill-Burton Bill, the adoption of the prepayment medical plans and methods of attracting younger men to rural areas. Most of the points that have been brought out in earlier meetings came out again in these discussions.

The meetings was adjourned with recommendation that a third meeting be held next year.

ANNUAL CONFERENCE ON MEDICAL EDUCATION

Dr. H. G. Weiskotten, chairman, gave some interesting points on current problems in medical education.

RESIDENCY PROGRAMS IN VETERANS HOSPITALS

Dr. Loren R. Chandler, San Francisco, Dean, Stanford University School of Medicine, acted as moderator.

Dr. Paul B. Magnuson, Washington, D. C., Acting Assistant Medical Director for Research and Education, Veterans Administration, went into considerable detail relative to the way the Veterans Administration is trying to cooperate with civilians and civilian medical schools and civilian doctors in carrying out the care of the veterans. He stated that the Veterans Administration is attempting to get the closest cooperation from all concerned for the best care of the disabled veteran. He stated that he had had 100 per cent cooperation from Washington and almost 100 per cent cooperation from the medical schools and civilian doctors themselves.

Dr. Harold S. Diehl from Minneapolis, Dean, University of Minnesota Medical School, explained how the training of residencies in the University of Minnesota is worked out.

Dr. Tinsley R. Harrison, Professor of Internal Medicine, Southwestern Medical College, Dallas, Texas, stated that they have two Veteran Administration hospitals, one ten miles west and one thirty miles north, at McKinney, Texas.

Dr. J. Roscoe Miller, from Northwestern University Medical School, Chicago, Illinois, and Dr. Burrell O. Raulston, University of Southern California, spoke of the fine cooperation that they are getting from Washington and from residents in their respective schools.

MEDICINE AND NATIONAL SECURITY

Dr. Edward L. Bortz, Philadelphia, Chairman, Committee on National Emergency Medical Service, American Medical Association, brought out the following facts:

(1) That during World War II there were 65,000 doctors in the Armed Forces to take care of fifteen million personnel leaving approximately 100,000 to take care of one hundred twenty million civilians. Obviously, it left the civilian population improperly cared for, the home doctors overworked and many of the military physicians with nothing to do.
(2) National emergency to civilian needs should be considered along with military needs.
(3) Medical personnel should not be called to military service until actually needed.
(4) Young doctors are not attracted in the military services as a life's work but this should be encouraged.
    (a) By scholarships such as West Point and Annapolis.
    (b) Superior young men should be invited into the Armed Services.
    (c) Better training should be provided during peace years.
    (d) Administrative and clinical officers be studied thoroughly and used where most efficient.
(5) Selective Service should study the fatigue element under the accelerated program and there should be a cabinet officer for the health office.

Dr. Harvey Stone, Baltimore, John Hopkins University School of Medicine, brought out that the Surgeon General should have a cabinet position, that the armed forces should stop frequent rotations, that the assistance of selecting nurses through the Red Cross be done away with, that the right men should be put in the right places, distribute medical personnel only as to needs, stop giving medical men non-medical men's duties and try to attract and keep young progressive medical officers.

Dr. Harold C. Lueth, Omaha, Nebraska, Dean, University of Nebraska College of Medicine suggested:

(1) That in times of peace a classification of all natural resources including medical personnel should be gotten.
(2) Reorganization of procurement and assignment services.
(3) Improve the services by National Security Board.

ACADEMIC PERFORMANCE OF PREMEDICAL VETERANS

The afternoon session was opened by Dr. Roy R. Kracke, Birmingham, Alabama, Dean, Medical College of Alabama, with the following observations:

(1) Students are three and one-half years older.
(2) Forty per cent are married.
(3) Studies are harder.
(4) Desire to get out as early as possible.
(5) Preferences are the following:
    (a) Engineering
    (b) Business
    (c) Premedical
(6) Much better student than before he went into the war.

THE HOSPITAL SURVEY AND CONSTRUCTION ACT

Dr. Thomas Parran, Washington, D. C., Surgeon General, United States Public Health Service, gave some interesting remarks. He stated that the act provides for the following:

(1) Survey
(2) Planning
(3) Construction
(4) Decentralization
(5) Consumer participation

He stated that there should be a Federal Hospital Council, composed of eight members, a State Hospital Council and an Advisory Committee. He stated that forty-seven states and territories had already started their surveys.

Building hospitals is determined by per capita income in each state. Funds are available for master state plan only, for general, tubercular, orthopedic and other special hospitals but not for domiciliary care. It provides for laboratory, outpatients, x-ray, nurses homes and other related facilities and plans for medical, nursing and technical help.

Increased need for more medical care:

(1) Sixteen per cent of all population is now over fifty-five years old.
(2) Fifty-two hundred medical graduates per year with 3,600 deaths will give us only a net gain of 7,000 doctors by 1960.
(3) Sixteen thousand doctors will be needed by Army, Navy and Public Health Services.
(4) Better financial status will increase the desire for medical attention.
(5) Estimated that there would be a deficit of 30,000 physicians by 1960, a deficit of 90,000 dentists, many thousand nurses and possibly a deficit in other categories such as technicians, etc.

Dr. Graham Davis, Battle Creek, Michigan, Director, Division of Hospitals, W. K. Kellogg Foundation; Member, Federal Hospital Council, in discussing Dr. Parran's paper said that this was not a plan but a program, that while poor states could get more money that they had to raise more money to meet the plan. He suggested that there be a combination of hospital and prepayment plans wherever this is feasible.

RESPONSIBILITIES OF A MEDICAL SCHOOL TO A COMMUNITY

Dr. James A. Greene, Houston, Texas, Professor of Medicine, Baylor University College of Medicine, went into the many various responsibilities of the medical school to the community. It serves:

(1) Undergraduate training.
(2) Research.
(3) Graduate training.
(4) Post-graduate training.
(5) Selects medical personnel in different communities.

MEDICAL APPLICATIONS OF ATOMIC ENERGY

Dr. Hymer L. Friedell, Cleveland, Director, Department of Radiology, University Hospitals, Western Reserve University School of Medicine, gave an interesting ultra-scientific talk of atomic energy in relation to future medicine particularly along the line of radiology.

Dr. Friedell's paper was discussed very ably by Dr. Raymond E. Zirkle, Chicago, Director, Institute of Radiobiology and Diophysics, University of Chicago.

At the dinner Dr. Andrew C. Ivy, Chicago, Vice-President, Chicago Professional Colleges, University of Illinois, gave a very interesting discourse on the Nazi War Crimes of a Medical Nature. He mentioned all of the crimes that we have read about in newspapers, many of which were carried on under the name of medical research but which are only medical torture.

FEDERATION OF STATE MEDICAL BOARDS

Presidential address by Dr. N. D. Buie, was read by Dr. Connally from Waco, Texas, because Dr. Buie was ill. He enumerated the advances that had been made in state medical licensure in Texas during the past forty-six years.

FOREIGN MEDICAL GRADUATES

Dr. Victor Johnson, Chicago, Secretary, Council on Medical Education and Hospitals, American Medical Association, stated that since the war began in 1940-41 it was impossible to determine what the status of the students in foreign medical schools have been, what type work they are doing, whether they have actually graduated from the school they claimed to be graduated from or the nature of their training. He suggested the following points in the consideration of foreign graduates:

(1) That only credentials sent directly from the schools should be considered.
(2) He should meet certain entrance requirements to medical schools.
(3) The student should be examined only by men who teach those subjects.
(4) Only two years of medical credit should be given.
(5) Freshmen should have at least one year premedical work before they enter medical school in this country.

He stated that we should be sure that his graduation is authentic, citizenship should be required, he should pass one of the State Boards or the National Board, that his residency should be in this country and admitted to the County Society in which he is living.

In 1945, 475 foreign graduates took board examinations in the United States, 345 of those took them in New York. He stated that many times the United States Department of Education can help in determining the quality of the schools from which the student graduates and the fact that his credentials are authentic. He warned that we should beware of faked medical credentials.

MEDICAL LICENSURE IN CALIFORNIA

Dr. Frederick N. Scatena, Sacramento, California, Secretary, California Board of Medical Examiners, stated that four methods are used for obtaining licensure in California:

(1) Examination.
(2) Commission in the Medical Corps of the Armed Services.
(3) Reciprocity.
(4) Diplomate of National Board.

He stated that foreign graduates have to be citizens of this country, must be graduates from approved schools and either spend their fourth year in California or have one year of rotation internship before they can be licensed. He stated that they have had two invasions of California by medical men and have increased the medical men 1,400, that they have 16,500 licensed to practice in California, 2,000 of whom are inactive and 1,000 still in the Armed Forces. He stated that their examinations consist of two parts, written and an oral. They are able to examine about 156 per day, that their examinations are unusually stiff and before a doctor should move to California to practice medicine he should make sure he will be allowed to practice in that state before he moves out there.

CONDUCT AND VALUE OF PRACTICAL EXAMINATIONS IN MEDICAL LICENSURE

Dr. Edwin S. Hamilton, Kankakee, Illinois, Member, Professional Examining Committee, Illinois Department of Registration and Education, was absent and his paper was read by Dr. Josiah Moore. He stated that their practical groups are made up in four sections, one-hour practical examination on each section:

(1) Surgical.
(2) Medical.
(3) E. E. N. T.
(4) Pathological and Clinical Laboratories.

He stated that a total of 300 points is required on all or an average of 75 per cent and that their failures are about 10 per cent whereas California's ranges from 40 to 60 per cent. The written examination is given on ten subjects with an average of 75 required and no grade must be below 60. His opinion was that the two examinations are very satisfactory in determining whether or not a man should be licensed.

A PROPOSED UNIFORM BASIC SCIENCE LAW

Dr. Orin E. Madison, Detroit, President, American Association of Basic Science Boards, enumerated the basic sciences as:

(1) Anatomy.
(2) Physiology.
(3) Chemistry.
(4) Bacteriology.
(5) Pathology.
(6) Pharmacology.

In his opinion, a Ph.D., man who had majored in certain subjects is probably better qualified to teach medical students than an M. D. who has not majored in it. He outlined in considerable detail the typical basic science law that should be passed in each state and recommended that the examination be given by basic science men who are not in its practice in any form—that each examination cover a two-hour period and that two days be required to give the examination. Then he ended what otherwise would have been a good paper by advocating that M. D.'s get together with osteopaths and chiropractors and work out a program that is agreeable to all.

RECENT LEGISLATION AFFECTING MEDICAL LICENSURE

Mr. J. W. Holloway, Jr., Chicago, Director, Bureau of Legal Medicine and Legislation, American Medical Association, mentioned a number of laws that have been passed in the different states. One stood out in particular, a Tennessee law, that stops all naturopaths, osteopaths, and chiropractors from practicing in the state. Texas has a new bill proposed which redefines the medical practice act that would preclude osteopaths on their examining boards and limits chiropractors and naturopaths to drugless therapy alone.

PRESENT DAY TRENDS IN MEDICAL LICENSURE

In the afternoon a panel discussion was held. It was brought out that there is no list of approved foreign schools and that the National Board in New York which examines most of these foreign doctors accepts no foreign graduate who graduated since 1942. Some brought out the fact that the basic sciences should be taught by an instructor who holds an M. D. degree because he understands better the viewpoints of the physicians and considerable exception was taken to the portion of Dr. Madison's paper in which he advised M. D.'s to sit down together with osteopaths and chiropractors.

NATIONAL CONFERENCE ON MEDICAL SERVICE

The meeting was called to order by Dr. Cleon A. Nafe, Indianapolis, Indiana.

Dr. Joseph S. Lawrence, Council on Medical Service, American Medical Association, Washington, D. C., discussed plans of the American Medical Association for medical service particularly in rural areas.

Dr. Herman E. Hilleboe, United States Public Health Service, Washington, D. C., explained in detail Public Law #725 commonly known as the Hill-Burton Hospital Bill. He stated:

(1) That finding the needs of the country was of first importance.
  (a) That the bill provides $3,000,000 for master survey of every state in the Union to find out where hospitals are needed and can be placed to the greatest advantage.
  (b) That this survey should be carried out under a state council, organized in each state with the help of the public health department wherever necessary.
  (c) It also provides for $75,000,000 per year for five years for construction needs.

(2) That hospital building is provided for on the basis of one to two. That is, the Federal Government will supply one-third of the cost and the state and community will pay the other two-thirds.

(3) That all types of hospitals are provided for, such as, tubercular, orthopedic, obstetrical and other special types of hospitals but that no provision is made for domiciliary care.

(4) Provision is made for the building of new hospitals, repair of old hospitals already built and construction of additions to hospitals now in existence.

(5) That is strictly a grant in aid.

(6) That it is not a federal project but strictly a state or local one and the initiation of the project must be started with the community after the national plan of the state made by a survey of the state hospital council has been made. The United States Public Health Service only guides and directs and in no way dominates either the construction of the hospital or the operation after it has been constructed.

VETERANS CARE: WHERE DO WE GO FROM HERE?

Dr. Paul B. Magnuson, Veterans Administration, Washington, D. C. made an extended statement at the beginning of his discussion about the plans and purposes of General Hawley and General Bradley in the Veterans Administration's care of disabled veterans. He stated that General Bradley has turned the job of medical care entirely over to General Hawley and that General Hawley has emphasized one plan for the care of the veteran which stood out in every endeavor which they were attempting. That is, what is best for the veteran? He stated that all red tape would be cut, most reports done away with in getting the best job done by the best people in the best way in the shortest length of time.

The following factors were emphasized:

(1) Keeping A. M. A. principles in medical practice.

(2) There are too many specialties. What the country needs and the veterans need in particular, are more practitioners of medicine.

(3) Physiologists are needed as well as pathologists.

(4) Didactic courses are not appreciable in hospital training but bedside training is important.

(5) Veterans hospitals need help from the civilian physicians.

MEDICAL COOPERATIVES

Mr. L. S. Kleinschmidt, Council on Medical Service, American Medical Association, Chicago, Illinois, brought out the following:

(1) That farm groups are now working out their own programs—compulsory insurance of any type is not acceptable.

(2) That there is a general cooperative movement among all farm groups, medical, housing, et cetera.

(3) That there are 15,000 of these cooperative groups, 7,000 consumers and 8,000 producers and that three-fourths of all of them are rural in nature; they have eleven million families of consumers served and eight million families of producers — an average of 4.8 per family. These figures represent some duplication.

(4) That the organization starts with the local group, then there is a regional group and finally a national cooperative congress which elects a director and sets the policies of the cooperatives.

(5) The A. M. A. and this Council has approved the cooperative health movement.

DEVELOPMENTS IN THE COUNCIL ON MEDICAL SERVICE

Mr. Thomas A. Hendricks, Council on Medical Service, American Medical Association, Chicago, Illinois, brought out the following:

(1) We need more publicity about what the medical profession in general is doing for medical services. We should tell the whole world about it.

(2) We should act now in all of our plans and not wait for the future.

(3) Forty-six states are either working on or have already adopted some sort of prepayment medical plan. Fifty-two plans have already been approved by the A. M. A. Council on Medical Services.

(4) How shall the association medical care plan function? He then produced two charts illustrating the inner relationship from the House of Delegates, through the Board of Trustees, the Council of Medical Service, all the way down through the State and County Medical Societies and the various councils of the A. M. A. and inner relationships.

At the panel discussion that afternoon many thoughts were brought out:

(1) That the general practitioner should be given his rightful place on the hospital staff.

(2) That the Board of Directors be appointed from the hospital staff itself.

Dr. Creighton Barker, New Haven, Connecticut, brought out that we are not producing enough doctors because many cannot afford to study medicine because of financial reasons.

Dr. Duncan W. Clark, Associate Dean, Long Island College of Medicine, Brooklyn, New York, emphasized the enormous trend toward specialization of the medical students and interns at the present time. He stated that at the present time there are graduated about 5,400 doctors a year and 3,600 are lost by death, that there are a few gained by foreign graduates. He suggested the following for the encouragement of more doctors:

(1) Grants and aids such as scholarships.

(2) That women be encouraged to go to medical schools.

(3) That the quality of medical teaching should be kept up and improved wherever possible.

(4) That many doctors are not in active practice due to advanced age or other disability.

(5) That group practice be encouraged where possible in order to give better type of care.

(6) That prepayment medical plans be encouraged by groups.

Dr. Darley, Dean of the University of Colorado, stated that there should be developed an academy or college of general practitioners and that the A. M. A. might create a specialty board along those lines. He stated that their college was sponsoring such residencies and that they had seven now taking that work and would have twelve by August. He brought out the enormous sacrifice that the men are making in developing specialties.

Dr. Leo G. Christian, Vice-Chief of Staff, St. Lawrence Hospital, Lansing, Michigan, stressed the importance of general practice in the hospital and stated that more doctors and less specialists are definitely needed. He stated that in our fight on socialized medicine we have to depend upon the general practitioner to win the fight.

Dr. Thomas P. Murdock, Chief of Staff, Meriden Hospital, Meriden, Connecticut, gave a program for the organization of a hospital:

(1) The Chief or Director of the Hospital.

(2) The Vice or Assistant Director.

(3) Chief of Surgery.

(4) Chief of Medicine.

(5) Chief of Obstetrics.

(6) Possibly others.

He stated that the program committee was possibly the most important committee, that ward rounds and conferences should be held regularly and that a well regulated hospital should have a pathologist, radiologist and anesthetist. Heads of the various departments should be certified men but this action should not be retroactive. A junior member should serve for five to six years and then be promoted to senior. The governing board should be purely local.

BOOK REVIEWS

Dentistry: An Agency of Health Service: By Malcolm Wallace Carr, D. D. S. New York, Commonwealth Fund, 1946. Pp. 216. Price $1.50.

This book was sponsored and subsidized by the New York Academy of Medicine. It presents, in an orderly fashion, a panoramic view of the development and progress of dentistry in America up to the present time and points out some of the needs for future development in this important field of health service. The author being a dentist (oral surgeon), the entire work presents the subject from the dentist's viewpoint.

Chapter VI, Hospital Dental Service, deals at some length with this somewhat more recent interest and activity of dentistry, and suggests the organization, duties and functions of the department. Several of these sound almost ideal, if they could be carried out. For instance (p.96) "not only should the patient's mouth be in a hygienic state but also the mouths of all who attend the patient—the surgeon, the anethetist, the nurses, the interns." Again, (p. 100) it is suggested that applicants for admission to the school of nursing should be properly examined (dentally) and the recommendations for treatment should be carried out before they are accepted. All this would be fine as far as it goes, if it can be done.

Three chapters dealing with research discuss the need for financial support, some problems, and the progress that is being made on them, and finally research in dental materials.

Dentists, physicians, and others will be interested in the general information relative to the organization of the profession of dentistry and the present, favorable position (mostly as part of universities) of dental education.

C. C. Bass, M. D.

Electrocardiography Including an Atlas of Electrocardiograms: By Louis N. Katz, A. B., M .A., M. D., F. A. C. P. (Second edition thoroughly revised with 525 engravings, including over 1,000 electrocardiograms). Philadelphia, Lea & Febiger, 1946. Pp. 824. Price $12.00.

This second edition, like the first, is divided into three sections. There are 824 pages including the bibliography. In the first edition there were 540 pages.

In the first section of the book there are descriptions of galvanometers, different leads and electrical principles as applied to the electrocardiogram and its place in practice.

In the first edition the author employed two chest leads. In this edition he has added CF 5. In other words he now uses three chest leads—CF 2, CF 4 and CF 5. It is believed that most cardiologists would recommend more than three chest leads. In some cases additional information can be obtained by moving the electrode farther to the right or to the left or even above these positions.

In section two, Chapter VI, he describes separately normal P waves, PR intervals, QRS complexes, ST segments, the T wave and the normal U wave. The effect of posture and age and its influence is mentioned. Most of these reproductions are good. There are tables throughout the book showing the salient features of normal and abnormal deflections. This should be of some benefit to the beginner.

In Chapter VII, he begins the descriptions of abnormal electrocardiograms. He advises that an attempt be made to place each electrocardiogram into a definite pattern, namely—coronary, heart strain, digitalis, acute diffused pericarditis, acute cor pulmonale, congenital heart, acute nephritis, dextrocardia, rheumatic mitral and chronic cor pulmonale. While it is not possible to put each abnormal tracing into one of these patterns it is well to keep them in mind. He justly warns of the hazard of attempting a prognosis or the clinical status of the patient on the electrocardiogram alone. It is only one part of the evidence that is to be fitted into the entire picture and the final clinical evaluation and prognosis must come from experience and common sense. This is well worth remembering.

He advises that the term axis deviation be dropped and in its stead two other terms used—axis shift and heart strain; the former meaning changes in the QRS direction caused by position and the latter due to strain or ventricular hypertrophy. One may have doubts about these terms clarifying a given situation.

In discussing the difficulties in differentiating the deep Q 3 seen in some cases of left axis deviation from one due to coronary disease no mention is made of the use of a left leg lead.

He calls attention to the value of the chest leads in left heart strain and also the possibility of the electrocardiogram being the first to show left ventricular hypertrophy. It is beginning to be realized that the electrocardiogram is probably the most sensitive indicator of ventricular enlargement or strain.

Chapter X is devoted to general considerations of coronary insufficiency and the electrocardiogram of coronary disease. It is the author's opinion that none of the tests, such as anoxemia, exercise, and cold, are of any consistent value in practice. It is very probable that this opinion will become more prevalent as time goes on.

Chapter XI is devoted to the electrocardiogram in recent myocardial infarction. The evolution of the different types are shown by reproductions of the electrocardiograms from patients with full descriptions of each curve.

In the last part of section two he describes the electrocardiogram in transitory coronary insufficiency, chronic coronary insufficiency, progressive and non-progressive and the electrocardiogram seen in pulmonary embolism, chronic lung disease, hypo and hyperthyroidism, valvular diseases, vitamin deficiency, hypertensión, chronic pericarditis, and so on.

Section three contains a systematic description of the electrocardiogram in the arrhythmias. There are numerous reproductions of curves. Most of them are good. The student can learn from this section much that is valuable, such as the differences between passive and active ectopic rhythms, and the difference between interference, disassociation and A. V. block.

The author believes that it is best to avoid the term bundle branch block and would put these cases in the following types — A common type — B uncommon type — CS type — D indeterminate type — one concordant — two focal — three miscellaneous. It would seem that this complicates the situation unnecessarily and that the terms left bundle branch block and right bundle branch block are justifiable. Each one may show different forms due to the position of the heart in the chest and myocardial changes.

Some of the author's views on electrocardiography are a little different to most cardiologists. He does not subscribe to the equilateral triangular hypothesis or vector analysis. However, anyone interested in electrocardiography will profit by reading this book.

J. M. BAMBER, M. D.

Exercises in Electrocardiographic Interpretation: By Louis N. Katz, A. B., M. A., M. D., F. A. C. P. (Second edition thoroughly revised, illustrated with 141 engravings, containing 166 electrocardiograms). Philadelphia, Lea & Febiger, 1946. Pp. 288. Price $6.00.

This edition follows the general plan of the first. The number of cases for interpretation has been increased to one hundred. The plan of presentation and most of the reproductions are good. The electrocardiogram is on one page and the description, interpretation, clinical story and correlation on the opposite page.

There may be some differences of opinion as to nomenclature but one should not hesitate to recommend this book to any one interested in electrocardiography.

J. M. BAMBER, M. D.

Principles in the Roentgen Study of the Chest: By William Snow, M. D. Springfield, Illinois, Charles C. Thomas, 1946. Pp. 414, 500 Illus. Price $10.00.

Although not so stated specifically in the preface, this book was presumably intended more as an aid to the clinician than to the practising roentgenologist. Starting with the very sound premise that radiology is more that the interpretation of shadows, Dr. Snow attempts to portray the roentgenogram as a means of correllating the physical, physiologic, anatomic and bacteriologic data of value in establishing a diagnosis. Unfortunately this laudable approach to the problem is marred by some poor reproductions of roentgenograms. Only too often there are uninformative and fail to substantiate what the author wished to emphasize. The section on technic is probably not adequate for most practising physicians; photofluorography is dispensed with in one paragraph while there is no mention at all of fluoroscopy. On several occasions, there is an attempt at clinical interpretation of the progress of a given disease; ordinarily this calls for detailed analyses which are not evident in the text. Many clinicians will take exceptions to some of the theories upon which Dr. Snow has based his clinical diagnosis in some of the cases chosen for illustration.

Because the basic idea of the book is good, and because there is a great need for such a book, it is to be regretted that these defects will keep this work from being of maximum usefulness to the practitioner.

SYDNEY JACOBS, M. D.

Clinical Biochemistry: By Abraham Cantarow, M. D., Max Trumper, Ph.D., 3rd ed., rev. Philadelphia, W. B. Saunders Company, 1945. Pp. 647. Price $6.50.

In the Preface the authors state that "the aim of this treatise is to consider how the internal environment of the body is altered by certain specific changes in tissue and organ physiology. It is further intended to indicate the manner in which the physician may best avail himself of information which can be obtained by biochemical studies. To this end the subject of functional diagnosis by chemical methods has been considered in considerable detail. With few exceptions the technic of the laboratory methods has not been discussed. The discussion has been restricted to those phases of biochemistry which are concerned with problems commonly encountered in clinical medicine and, therefore, purely abstract and theoretical considerations have been excluded."

Progress in biochemistry since the last edition (1939) of this standard work has necessitated extensive supplementation and revision of material previously presented. The present book is complete, concise, accurate and up-to-date.

A. O. KASTLER, Ph.D.

Pathology of Tropical Diseases: *An Atlas*: By J. E. Ash, Colonel, M. C., U. S. A. and Sophie Spitz, M. D., C. S., A. U. S. Philadelphia, W. B. Saunders Company, 1945. Pp. 350. Price $8.00.

Prior to the emission of this book, there did not exist in English, a single volume devoted solely to the pathology of tropical diseases. The need and demand for one has been steadily growing for the past two decades and with the war's ramifications this need became acute. Unavoidable delays postponed the publication for almost two years.

As the title indicates the work is an Atlas and is primarily a collection of superb photographs and photomicrographs, some in color, of the chief diseases encountered in the tropics. Maps showing distribution and schematic drawings giving the life histories are supplemented by brief but adequate descriptions of the clinical and pathological findings. Thus the brief descriptive writing does not pretend to be an exhaustive treatise on all the tropical conditions discussed but serves well to orient a reader not already familiar with these phases.

An early review in a leading journal singled out a number of statements as inaccuracies which are definitely not when read into the context. "Chi-goes" is used on page 325 with harvest mites but the correct explanation is given on the following page. There can be no excuse however for the title of two pages of illustrations as "Chigoe Bite." Conversely, none of the usual texts mention the important fact that febrile states are predisposing factors in the migration of adult ascaris. Moreover, the sketches of the type insect in the lower corner of the maps showing distribution are entomologically poor but adequate to show the class of arthropod involved. It is well known that transplacental malaria infection, through the *intact* placenta does not occur. With Leishmaniasis, it is noted that the successful biting experiments have not been mentioned.

In conclusion, it may be stated that the work fulfills admirably the role it has undertaken and can be recommended without hesitation to those requiring a reference work of this kind.

A. J. WALKER, M. D.

Modern Urology for Nurses: By Sheila Maureen Dwyer, R. N., B. S. and George W. Fish, M. D. Philadelphia, Lea and Febiger, 1945. Pp. 287, illus. Price $3.25.

The authors have presented a text dealing with the care of urologic patients in a centralized urologic service planned and equipped similar to that of the Squier Clinic, Presbyterian Hospital, Columbia University Medical Center, New York City. The text is of value to not only the nurses but the entire urologic personnel.

MONROE WOLF, M. D.

A Surgeon Looks at Life: By Richard A. Leonardo, M. D., Ch.M., F. I. C. S. New York, Froben Press, 1945. Pp. 128. Price $2.00.

This is a book of essays dealing with a variety of loosely related subjects such as faith cures, psychology in medicine, birth control, sterilization, suicide, euthanasia, heredity and environment, juvenile delinquency, geriatrics, life after death, and the rule for a good life. Every subject is approached from the philosophic aspect, and usually against a historical background. The science and art of surgery, however, are barely mentioned, and in many of the essays there is not very much medicine.

Readers interested in reflections on the subjects listed will find the book easy reading, though not very deep. So little of the writer's surgical experience appears in it, however, that the title is rather misleading.

FREDERICK FITZHERBERT BOYCE, M. D.

Practical Malariology: Medical Manual, National Research Council: By P. F. Russell, M. D., M. P. H., L. S. West, Ph.D., and R. D. Manwell, Sc.D. Philadelphia, W. B. Saunders Co., 1946. Pp. 684. Price $8.00.

This recent addition to the literature on malaria presents an excellent coverage of the various phases of practical or applied malariology. It is highly recommended for the many individuals interested in this subject and particularly for workers engaged in field survey and control, in the laboratory and in the teaching of malariology. The authors have selected from and evaluated the voluminous published material relating to malariology based upon their considerable experience. The clinical features of malaria appear to be somewhat subordinated to the field and laboratory phases of malariology, and some aspects of the biology of the plasmodia are touched upon lightly. This is probably justified by the purpose of the book and by the importance of entomology, parasitology, laboratory diagnosis and sanitary engineering in the prevention and control of the disease. Much space is devoted to malaria survey and control, from which the reader may profit from the wealth of experience and contributions of the senior author in malaria control in various parts of the globe.

The important new developments relating to malaria, particularly the products of the intensive wartime research program, are presented. Some of these include the recent additions to the armamentarium of antimalarials, such as chloroquine and paludrine, and the improvement in the methods of using atabrine for the suppression and treatment of malaria. Information on pentaquine apparently had not been released for publication at the time of printing. Other recent developments, as DDT, its remarkable significance and uses in malaria control, its varied methods of dispersal with ground equipment or by airplane, the improved mosquito repellents, and the insecticide aerosol dispenser, are considered. Lessons learned in military malaria control from the programs and field experiences of the armed forces are drawn upon fully. A few of these are the experimental studies on malaria suppression with atabrine, new malaria control technics and military organization for effective malaria control. The recent advances in the technic of cultivation of malaria plasmodia receive deserved consideration. Worthy of mention is the treatment of the history of malaria which is presented in a most readable style.

The volume contains 238 figures and is printed on heavy gloss-finish paper. There is an appendix consisting of keys to world anophelines. This practical book merits wide distribution and can be read with great profit by those engaged or professionally interested in malariology.

J. C. SWARTZWELDER, M. D.

Gastroenterology in General Practice: By Louis Pelner, M. D., with the collaboration of Louis Held, M. D., and contributions from Alexander Lewitan, M. D., Sanuel Waldman, M. D., and Siegfried W. Westing, M. D. Springfield, Ill., Chas. C. Thomas Co., 1946. Pp. 285, illus. Price $7.50.

In this text the author discusses the commoner derangements of the gastrointestinal tract at a practical level of value to the general practitioner.

After a review of history taking follows a series of chapters covering the various specific gastroenterological entites including enteric allergy and hyperinsulinism. Then come four chapters on special diagnostic procedures including a discussion of gastroscopy and two well-illustrated chapters on radiography. The closing chapters are devoted to such subjects as vitamin deficiencies, abdominal symptoms of cardiac disease, pre- and postoperative nutritional management, and the psychosomatic aspects of the subject.

The small size of the text seriously limits its value, in this reviewer's opinion; it offers not much more intensive coverage than the corresponding sections of the standard textbooks of medicine, and as a reference source, is too limited in detail to offer aid in solving a specifically encountered problem. Parenchymal liver disease is disposed of in a scant 7 pages, and the discussion of hepatic physiology is too superficial to be of much value. Little attention is paid to the application of fundamental physiologic principles, and the wisdom of segregating psychosomatic implications instead of weaving them into the discussion of specific diseases is questionable.

Despite these criticisms, the author has produced a practical, readable, well-illustrated text which should be of real value to general practitioners seeking a review, and to students interested in a concise survey of the commoner diseases of the gastrointestinal tract.

J. R. Snavely, M. D.

Clinical Methods in Neuro-Ophthalmological Examination: By Alfred Kestenbaum, M. D. New York, Grune and Stratton, 1946. Pp. 400, illus. Price $6.75.

Although bound in fabricoid and printed on thick rough paper, this volume of approximately 400 pages contains a wealth of useful information and an understanding of which any author could be deservingly proud. Aside from being a valuable book of reference and instruction, it will also serve the purpose of helping both ophthalmologists and neurologists see neuro-ophthalmic problems from a unified point of view, which unfortunately has not been the case during the past two decades. The subject is presented in an orderly manner, and complex problems are greatly simplified. The anatomy and the diagnostic problems involved by the numerous groups of visual and pupillo-motor fibers and impulses from the retina to the visual motor brain and the ultimate motor fibers are described with great clarity. Many of the examination methods mentioned have not been previously presented in current books on ophthalmology, especially those published in this country. The illustrations consist of about 65 line drawings which have been exceptionally well chosen; 25 tables and numerous summaries further help to differentiate somewhat similar conditions. The bibliography contains about 500 references, each with the page number used in this volume. A 12 page glossary will be of especial value to those who are not familiar with neuro-ophthalmic terminology. The 12 page index is accurate and complete. The neuro-ophthalmic examinations suggested are reasonably complete, but apparently could have been arranged in a more efficient manner from an ophthalmologist's point of view, and the table of contents could have been amplified to advantage.

Chas. A. Bahn, M. D.

Clinical Hematology: By Maxwell M. Wintrobe, M. D., Ph.D. 2nd ed. rev. Philadelphia, Lea and Febiger, 1946. Pp. 862. Price $11.00.

It is a pleasure to be in a position to review the second edition of Wintrobe's *Clinical Hematology*. The first edition, which might be spoken of as an offspring of Wintrobe's original contribution to Tice's Practice of Medicine, was almost full grown at its birth. The second edition which has been thoroughly revised might be likened to the full maturation of the red cell. It is a finished and complete book.

The second edition contains the many advances that have been made in the study of the blood, its genesis, its diseases and the diseases of the blood-making organs. The author has kept up so well with the current medical literature that, allowing for the time taken in printing the book, it very closely and in fact almost completely covers those discoveries that have been made and recognized as worth while up to the present minute. Of course some exceptions could be made to this broad statement. New information in regard to the Rh factor appears almost daily and it would be impossible for a book to contain every last bit of information containing the Rh factor and the same may be said of the relationship of macrocytic anemias to vitaminology.

In addition to almost the latest information in regard to the two subjects mentioned above, it might be said that Wintrobe has included in this new volume considerable data concerning other vitamins and the part that they play in hematopoiesis. The newer knowledge of the porphyrins has been incorporated as well as the knowledge of the important action of the nitrogen mustards in the treatment of Hodgkin's disease.

The second edition of this book has not only been "thoroughly revised", it has been almost completely revised. Many of the pages have been completely rewritten; there has been a large number of new illustrations added and some of these are colored plates. One completely new chapter has been added to the other edition of this work.

It is difficult to write about this book without expressing possibly too fulsome praise of its contents. It must be conceded, however, by any reader that the book is extremely well organized, the writing is clear, concise, succinct, and with the exception possibly of two or three illustrations, these are much to be praised.

J. H. MUSSER, M. D.

Electrocardiography in Practice: Ashton Graybiel, M. D. and Paul D. White, M. D. 2nd. ed. Philadelphia, W. B. Saunders Co., 1946. Pp. 449, 323 illus. Price $7.00.

This book is divided into five parts with two appendices at the end. In the first part there is a short but adequate description of the normal electrocardiogram. The electrical axis based on the equilateral triangle hypothesis and the difference between the instantaneous electrical axis and the mean electrical axis as used in clinical electrocardiography is explained. A good description of the precordial leads and the influence of the distant or indifferent electrode on the form of the precordial curves are given.

The electrocardiograms reproduced in this book show precordial leads CF 2 and CF 4 and CF 5 when more than one precordial lead has been made. This is enough in most cases but it should be remembered that not infrequently points further to the right or to the left or above these points may give valuable aid.

Part two is devoted to the variations of the normal electrocardiogram. The importance of the position of the heart, the influence of the position of the patient, whether sitting up or lying down are presented.

Part three takes up the arrhythmias. After a short discussion there is a table summarizing some of the important points in diagnosis.

Part four consists of descriptions of etiological types and patterns. First auricular and ventricular strain, second localized lesions, myocardial infarcts, etc., third generalized cardiac involvment. There is a table summarizing the usual electrocardiographic findings, normal and abnormal, in various types of heart disease. The effects on the electrocardiograms of drugs and physiological processes are described.

Part five consists of electrocardiograms for practice interpretation. Throughout the book, except part one, the electrocardiogram is shown on the right hand page facing the reader and the interpretation on the curves, clinical findings and comment are on the opposite page. This is a good arrangement.

Most of the reproductions are good and well selected. However, if there had been more precordial leads and a greater number of cases showing more than three positions of the chest electrodes it would have increased the value of the book.

There are a few curves in which there may be a difference of opinion in the interpretations. In one—Fig. 291 on page 401—it is stated that the limb leads are within normal limits. There is left axis deviation with T 3 upright and larger than T 1. In the opinion of the reviewer such curves are not normal. The electrocardiogram shown in Fig. 292 on page 403 is intrepreted as being old anterior infarct with left bundle branch block and deep Q waves in CF 4 and CF 5. Q waves are not found in precordial leads from the left side in left bundle branch block unless there is infarct of the septum. The Q wave in Lead 1 is also unusual in left bundle branch block. This may be more or less academic in this case but it should be discussed. It migh have been better to have shown more curves with standardization and to have called attention to it from time to time.

The discussion of the clinical findings and the frequent reference to the management combined with the interpretations of the electrocardiogram adds to the value of the book and gives one a better perspective of the value of the electrocardiogram in clinical cardiology.

There are two appendices at the end of the book. Appendix one—unipolar limb leads and esophageal leads. The unipolar limb leads are used to determine the electrical position of the heart. VF is useful in the differential diagnosis of old posterior infarct. Appendix two shows the effect of exercise and anoxemia on the form of the electrocardiogram.

In the opinion of the reviewer this is one of the best books on this subject.

J. M. BAMBER, M. D.

The Washington Institute of Medicine announces that a limited number of sets of volume 1-3 of the Quarterly Review of Obstetrics and Gynecology are still available for puchase. Physicians interested should write to the Washington Institute of Medicine, 1720 M St., N. W. Washington, D. C.

PUBLICATIONS RECEIVED

W. B. Saunders Company, Philadelphia: Principles and Practice of Obstetrics by Joseph B. De Lee, M. D. and J. P. Greenhill, M. D.

Charles C. Thomas, Springfield, Ill.: Penicillin in Syphilis by Joseph Earle Moore, M. D.; a Handbook of Commonly Used Drugs by Michel Pijoan, B. A., M. D. and Clark Harvey Yeager, M. D., Dr. P. H.; Techniques and Procedures of Anesthesia by John Adriani, M. D.

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ANTHRAX IN LOUISIANA

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New Orleans

A patient with anthrax recently entered Charity Hospital at New Orleans. The patient stated he had first noted a painful rapidly progressing lesion on the right hand and had complained of it to a neighbor who asked if he had been skinning dead cattle. When he replied in the affirmative, the neighbor told him he had charbon and directed that he go immediately to his doctor who confirmed the diagnosis by microscopic examination, administered antiserum and had the patient hospitalized over one hundred miles from his home within 72 hours following the stated time of appearance of the cutaneous lesion. When asked if he had ever heard of anthrax or charbon before, the patient named three persons of his acquaintance who, he said, had previously had the disease and had recovered. He knew several farmers who had lost cattle from anthrax in the last few years. In one instance the veterinarian had called the disease anthrax after examining blood from the ear of the dead cow. He further stated that as he was leaving for the hospital his father was burning the hide and carcass of their cow with crude oil.

This situation caused me to consider what was the history and present status of anthrax in Louisiana to make it so well understood and recognized by both layman and physician and led to the following study of the disease.

It appears that anthrax has been prevalent in Louisiana from the days of its early settlement by the French. Carpenter[1] stated that the disease was first observed among the deer in the marshes along the Mississippi River and that soon thereafter it made yearly attacks on cattle in the Feliciana Parishes adjacent to the river. From those times the disease has managed to maintain itself among domestic animals in the state. Cattle, mules, horses and sheep, in the order listed, are the animals most frequently mentioned as contracting anthrax. The frequency of infection in each appears in direct ratio to the numbers of each species in the state. Hogs and dogs are less frequently mentioned, but statements do appear to the effect that these animals, feeding upon the carcasses of anthrax animals may develop the disease and die. Some of the largest epizootics are recorded for the periods between 1830-1850, 1895-1902 and 1920-25. The two largest recent outbreaks appeared in St. Bernard Parish in the fall of 1943 and in twelve of the north-central parishes in the summer of 1946. In connection with the latter, the Louisiana Livestock Sanitary Board reported[2] the loss of 2,000 farm animals and stated further that the disease was of the acute or fulminating intestinal type with no symptoms of illness being shown until a few minutes before the animal's death when convulsions occurred. The accompanying map gives a partial picture of the time of appearance and the distribution of epizootic anthrax in the state but makes no

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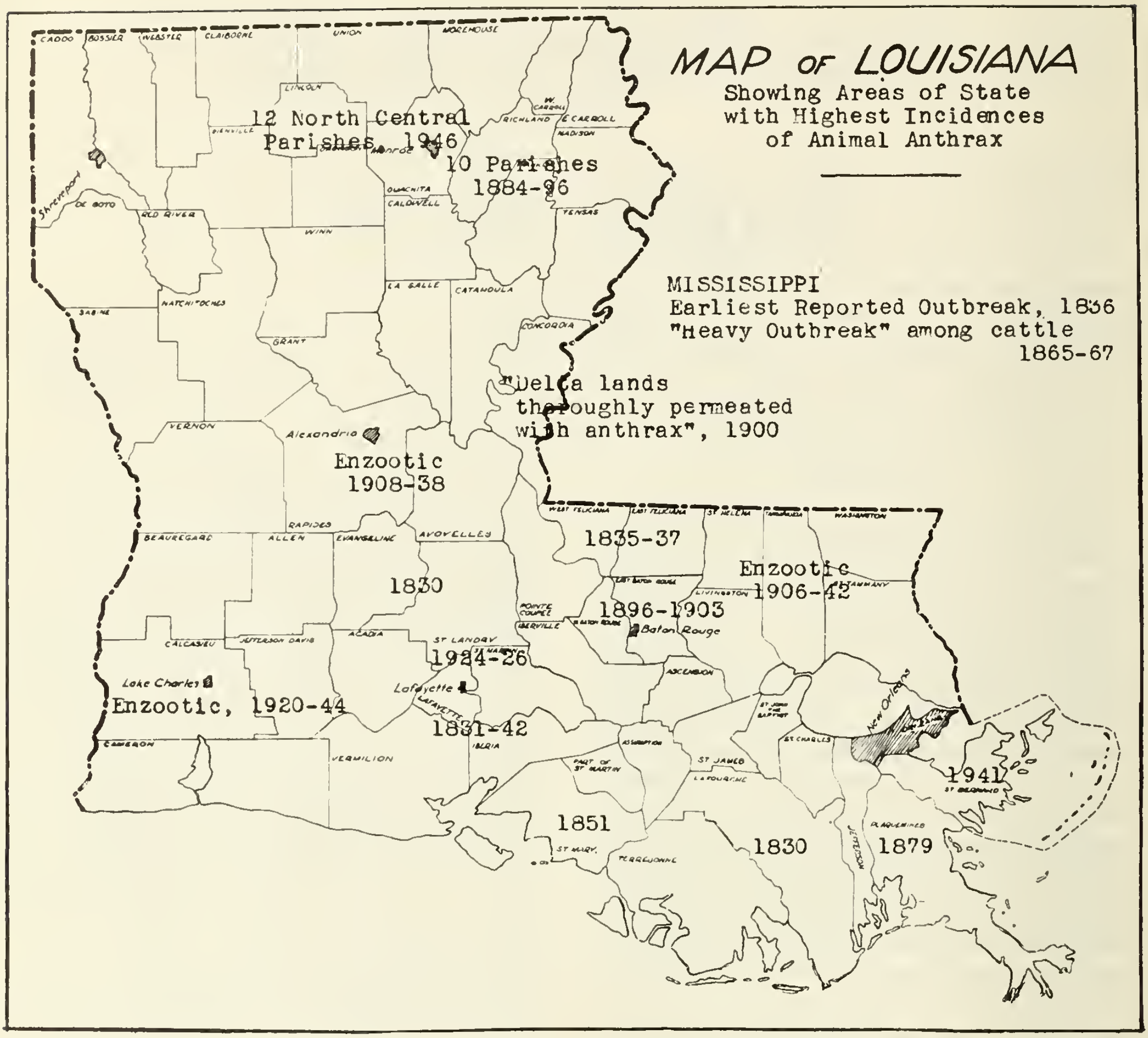

attempt to record small scale occurrence of the disease.

Animal immunization has been practiced extensively even before 1900 in the parishes in which the disease had been most prevalent. Archinard[3] stated in 1896 that a supply of vaccine for animals was always kept on hand in New Orleans. The State Agricultural Experiment Station reported in 1901[4] that over 30,000 immunizations with the Pasteur anti-anthrax lymph materials had been given during the course of one year. Annual immunizations are still continued in some of the central parishes where a few cases may be expected to occur each year in spite of the number of livestock immunized.[5]

Biting flies are more frequently mentioned by Louisiana writers as agents of anthrax transmission than by writers from other areas of the United States. Although mechanical transmission of this disease was not experimentally proved until the time of Mitzmain's studies[6] in 1914, it had been accepted in Louisiana as an important mode of transmission of the disease for at least 80 years prior to this time.[3, 4, 5, 7] The biting flies variously mentioned are the deer-fly, green-headed fly, horse-fly, grey biting-fly and the gad-fly, all of which belong to the same family, *Tabanidae*.[8] The females of this family are blood-sucking and accept a wide range of animal hosts. Their breeding places are in swamps, marshes or along river bottoms where they deposit their eggs near or over the water. Various writers have stated that anthrax appeared during or following periods of the summer or fall when great numbers of biting flies were present. Dalrymple[4] remarked upon the plague of such flies in 1901 when anthrax was appearing in high incidence near Baton Rouge. Baldridge[9] particularly called attention to the presence of flies in large numbers at the time he attended his cases.

As contrasted with the intestinal type of disease shown in animals inoculated by the ingestion of anthrax-infected forage, insect transmitted disease is characterized by the lesion appearing in or under the skin at the site of injection of the organisms. Since these skin tumors, nodules or carbuncle-like sores, as they are variously described by veterinarians and farmers, are very apparent and are approachable for treatment, more human cases have been reported from contact with cutaneous animal anthrax than for the intestinal type of the disease. This was especially true when veterinary therapy was directed primarily toward the cutaneous lesion and included such procedures as excision, blistering, cauterization or even setting fire to the lesion after covering it with oil. At present exposures are limited to contacts with animals not suspected of having anthrax. One of Louisiana's most recent animal contact cases is that of a veterinarian who developed anthrax after performing an autopsy on a dead bull.

Until such time as the present method of disposal of anthrax carcasses by burning or by deep burial with lime came into general use, the carcasses were either left to disintegrate where the animal had died or were dragged back into the swamps where flies might feed upon them or where surface waters might wash the infection over a wide range of territory. Furthermore, Louisiana's open range laws, permitting livestock to roam at will, have very effectively combined with other factors favoring anthrax transmission and thus, during the course of the years, large areas of the state have been seeded with the highly resistant spores of *B. anthracis*.

It appears that the first scientific report of human anthrax in Louisiana was made in 1830 by Ellison.[10] He described eight cases in the south and central portions of the state. One of them was a Spaniard who had eaten beef from an animal that had died; another, a man who had skinned a dead cow; and the remaining six were persons who had been bitten by insects. The first two of the patients died. Smith[11] reported attending cases in the same area of the state during the period from 1834-1843, but gives few details concerning his cases. Carpenter[1] described eight cases that occurred in the Feliciana Parishes during the years 1835-1837. These persons had either handled hides of farm animals that had died of anthrax or had been bitten by insects. Baldridge[9] in 1851 described a series of human cases associated with epizootic anthrax in St. Mary and Vermilion Parishes. While he definitely associated the biting fly with transmission of the disease, he stated that persons (drovers, shepherds and butchers) having most intimate contact with farm animals were most likely to contract the disease. In 1879 Fox[12] mentioned anthrax as a disease long established in Plaquemine Parish (along the lower stretches of the Mississippi River) and specifically stated that he had seen six cases of the disease in 1853. These cases were confined to the residents of one plantation. The first of them was the hostler who had cut strips from the hides of animals that had died of anthrax the previous spring. The remaining cases were among persons living in cabins adjacent to where the hides were stored.

Staples and Dalrymple,[13] discussing the 1896 epizootic in ten northern parishes (including Tensas, E. Carroll, Richland, Franklin and Madison), mentioned a human case in Madison Parish; that of a negro who had attended sick mules. They included Dr. Cordill's report of the 1884 anthrax attack in Franklin Parish, in which he said, ". . . and many people were affected, and some died from the disease. All of those living, who were attacked have scars to show for it."

Dabney's case,[14] that of a man who

"worked in a hide-store" in New Orleans, is one of the few cases reported in the medical literature during the last half of the nineteenth century. It has the distinction of being the first human case to appear in the yearly statistical reports (1898) of the state. Subsequent to 1900, when case reporting became less fragmentary and when mortality reports had become more complete and more nearly accurate, human anthrax cases have been recorded with definite regularity. However, the number of cases reported probably has never approached the real incidence level of the disease. For example, during the period of unusually high prevalence of the disease in the Evangeline Parishes (1920-1925) one physician treated more than sixty human cases of anthrax in a single year.[5] Of this number, a total of one case achieved entry into the state's statistical records. That incomplete case reporting still exists is evidenced by the 1945 reports of one case and two deaths. Table 1 gives the cases reported to the Louisiana State Department of Health for each year since 1920.

TABLE 1

Human Anthrax Cases Reported in Louisiana For the 26-Year Period, 1920-1945, Inclusive*

Year	Number of Cases	Year	Number of Cases
1920	6	1933	3
1921	4	1934	6
1922	4	1935	3
1923	18	1936	1
1924	11	1937	4
1925	1	1938	3
1926	9	1939	2
1927	3	1940	2
1928	6	1941	3
1929	2	1942	6
1930	6	1943	3
1931	3	1944	0
1932	3	1945	2
		1946	0†
		Total	114

* Taken from Communicable Disease Reports of the Louisiana State Department of Health.

† To November 1, 1946.

The comparative incidence of anthrax reported in Louisiana (based upon the summary of Smyth[15] and the U. S. Public Health Reports[16]) is shown in table 2. On a population basis it is evident that this state reports a comparatively high anthrax incidence. The states with higher frequencies are certain of the New England and Middle Atlantic states wherein the disease differs epidemiologically in that it appears among industrial workers handling imported hides, hair and wool. Table 3, drawn from sixty Louisiana case histories from which sufficient data could be obtained, shows that anthrax, as it has occurred locally, is almost entirely an agricultural disease rather than one of urban industry. While 13 urban dwellers with such occupations as electrician, clerk and chauffeur are included in the 60 cases, investigation reveals that all probably got their infection in rural areas. The clerk, for example, worked in a store in New Orleans but used part of the liver of a dead cow for bait during a four day fishing trip in Alabama and returned to New Orleans with the cutaneous lesion already evident and painful. The electrician, wiring a house in the country, suffered from numerous bites of "cattle flies" while at work. The cutaneous lesion appeared within 24 hours at the site of one

TABLE 2

Comparison of Numbers of Human Anthrax Cases Reported in the United States with Number of Cases Reported in Louisiana.

	United States		Louisiana	
Period	Number of Cases	Yearly Average	Number of Cases	Yearly Average
1919-1938	1,683	88	93	4.65
1938-1942	375	75	16	3.20

TABLE 3

Occupation in Relation to Exposure to Anthrax in Louisiana*

Occupation	Cases	Contact with animals or animal products	Primary lesion developing at site of fly bite	No history of contact with sick animals or of fly-bite
Rural				
Farmer or farm laborer	41	24	10†	7
Housewives	4	3	0	1
School	2	2	0	0
Urban				
City dwellers whose work or recreation took them to country ††	12	10‡	2‡	0
Worked in hide store	1	1	0	0
Totals	60	40	12	8

* Taken from records of the Charity Hospital at New Orleans, from Dabney,[14] Baldridge,[9] and from the Communicable Disease Reports of the Louisiana State Department of Health.

† Six of these cases also gave a history of definite contact with anthrax infected animals.

†† Group includes three laborers, one butcher, one blacksmith, one clerk, one carpenter, one fisherman, one chauffeur, and one electrician.

‡ Contacts occurred in rural areas.

of the bites. The chauffeur cut his hand while skinning cows that had died suddenly on the farm of a friend. The only obtainable case that is definitely connected with industrial processing of hides is Dabney's case previously mentioned. No data are given, however, as to whether the hides were domestic or imported. The only case wherein infection was probably obtained outside of Louisiana is that of the clerk mentioned above.

Table 4 compares the type of anthrax and the location of the cutaneous lesion for a series of 640 cases throughout the United States[15] with a smaller series of 44 cases cared for at Charity Hospital at New Orleans. While ocular, pulmonary and intestinal anthrax do occur in Louisiana, no patients have been admitted to Charity Hospital and few to any other hospital, as indicated by correspondence with 40 of the larger hospitals of the State. Physicians say that when they have been called to see these patients their reaction has been that since death appeared certain and imminent, the patient might better be left in his home. The location of the cutaneous lesions in the Louisiana cases may be influenced by the number of insect transmitted infections, in so far as the lesion may appear on any exposed body part. Certainly there is no large percentage of lesions on the back and shoulders as appear in persons carrying bales of infected hides.

TABLE 4

Location of Primary Lesion in Anthrax Cases

	640 Cases throughout United States 1919-1938	44 Cases Charity Hospital 1904-1944
Internal Anthrax		
Pulmonary	4	0
Pulmonary and intestinal	2	0
Intestinal	2	0
Meningeal	3	0
External Anthrax		
Face, neck and head	309	8
Upper extremity	264	26
Lower extremity	23	2
Chest, abdomen and back	23	3
Multiple lesions	9	5
Not stated	1	0

The only reference found regarding a second attack of anthrax in any person in Louisiana is that of the negro in Madison Parish who attended sick mules.[13] Hull[17] described four cases found in the literature and stated that in one instance the second attack occurred within a year following the first infection. No mention is made of the interval between the two attacks suffered by the Madison Parish negro. No reference has been found to establish person-to-person transmission in Louisiana although it is occasionally reported elsewhere.[17]

No proved cases of shaving brush anthrax have been found. The nearest approach is the case, recorded at Touro Infirmary at New Orleans, of a 49 year old white farmer who scraped the surface of a pimple on his neck one morning while shaving. A few hours later the pimple had become a large painful lump from which anthrax bacilli were obtained after admission to the hospital. The patient said that on the morning when he scraped the pimple he had used a new shaving brush and strop for the first time. Two days previously, however, he had examined a stray hog found dead on his property. Cultures from the shaving brush, stone, strop and mug, sent to the State Laboratories, were reported negative for anthrax bacilli. The hog was not examined, but since animal anthrax was present in the area at that time (W. Baton Rouge Parish, 1923), it appears to have been the probable source of the infection.

An unreported series of four related cases[5] demonstrates exposure to intestinal anthrax and the chances against its development. In 1924, a white farmer from Iberia Parish appeared at his physician's office with a four day old anthrax lesion on his lower arm. He said he had killed and skinned a cow that appeared to have been suffering from physical injury. He had dressed the carcass and sold the meat to neighboring families. Among 53 persons exposed either to the raw or cooked meat, three cases of anthrax developed. These were all of a cutaneous nature and were limited to those who had prepared the meat for the table. Billings[18] has said that proper pickling or thorough cooking will destroy anthrax organisms and cites the report of

200 persons who ate meat taken from a diseased ox. None of those who ate the cooked meat became ill, but five persons who handled the uncooked meat developed anthrax. While much Louisiana home-cured meat is salt pickled, it is not consumed without subsequent cooking. Furthermore, very little smoked meat of a variety to be consumed without cooking is processed in the rural areas of the state. The small amount of wool grown in the state and the absence of any large woolen or hair-processing industries has probably been responsible for holding pulmonary anthrax to similar low percentages of total anthrax cases.

Smyth[15] in 1941 reported an anthrax case fatality rate of 22 per cent for the United States for the period 1919-38 with 16 per cent case fatality for the last five years of the period. For the same time period Louisiana stood higher with 30 and 22 per cent case fatalities, respectively. Since that time anthrax deaths have decreased considerably. Lucchesi and Gildersleeve[19] reported treating 67 cases in Philadelphia without a fatality. Gold[20] reported 60 industrial cases treated between 1933 and 1942 with only one fatality. Tabershaw and Morris[21] reported a case fatality rate of 9.3 per cent (four of 43 cases) for Massachusetts for the period 1935-1942. While the Louisiana fatality rates are higher for reported cases, there is much reason to believe that these rates are quite inaccurate as only the more malignant type of case is reported. Louisiana physicians state that the cutaneous case is often not malignant, that it assumes the characteristics of the common carbuncle and that etiologic differentiation is ascertained only by microscopic examination of crusts from the lesion. Bell and Legge[22] in London in 1912 called attention to the frequency of this type of nonmalignant anthrax. Local physicians also state that if they see the patient before septicemia has occurred, they can care for him adequately in his own home. These cases are seldom reported.

Of 44 patients admitted to Charity Hospital from 1906 to date and with the diagnosis by laboratory findings, only five deaths have occurred. Two of these were in patients with the primary lesion on the face, two with multiple lesions on the arms, and the fifth with the cutaneous lesion in the area of the inguinal nodes. All five were admitted before the days of sulfonamide therapy. Four admitted since 1940 have responded to sufapyridine, sulfanilamide or sulfathiazole used in conjunction with tetanus antiserum.

The only Louisiana case history[23] obtainable in which penicillin was used was one of the two cases reported in 1945. This boy cut his foot on a piece of wire near a manure pile. Four days later, when the foot and ankle were swollen and one degree of fever was present, the patient was taken to a local physician because of the fear of tetanus. Four or five days later, upon admission to the hospital, the temperature was 101° and examination revealed a woody, brawny, painless induration of the entire leg. The diagnosis of anthrax septicemia was made when anthrax bacilli were found in the blood smear. An initial dose of 100,000 units of penicillin was given followed by 50,000 units every two hours. The child died 52 hours after admission to the hospital. While this patient did not respond favorably to penicillin, this failure is possibly due to the fact that treatment was begun late in the course of the disease on the eighth or ninth day when septicemia was well established. Had the case been recognized earlier, as usually happens in Louisiana, more favorable results might have been expected. Current literature, beginning to report favorable anthrax case responses to penicillin therapy, is concerned with cases in which diagnosis is made earlier and penicillin is given before septicemia has occurred. For example, Murphy, La Boccetta and Lockwood[24] reported prompt clinical response to penicillin in three cases of cutaneous anthrax seen on the third, fourth and fifth days, respectively, after appearance of the skin lesion and with blood cultures negative for *B. anthracis*.

The seasonality of human anthrax shows a pattern of case distribution throughout the year with a definite peak during midsummer (table 5). This seasonal case frequency in man parallels that of animals in the state.

Distribution of human cases in the parishes of the state is again similar to the distribution of the disease among animals with no reports of human cases found for certain of the north and northwestern parishes and with animal anthrax less frequently reported from the same areas. Anthrax is in many ways associated with the bayous, marshes or streams of the state. One writer states that as anthrax causes a high fever and an associated thirst for water, the sick animal goes to the bayou and dies there. Others point out the old practice of dragging the carcasses of all dead animals back into the swamps. Others have stated that anthrax first appears among cattle pastured in swampy territory. Others call attention to the frequent reversal of direction of water flow in a bayou with the result that infection may be carried either up- or down-stream. The northwestern section of the state, with less frequent appearance of anthrax, contains the highest lying land within the state where an elevation of 400 feet above sea-level is reached, receives less than the average yearly precipitation (57 inches) for the state, and, as a result, is better drained with less flooding or back-flow of surface waters. It appears that these features have been responsible for reduced anthrax incidence in the area.

TABLE 5

Seasonal Occurrence of 111 Cases of Anthrax in Louisiana
By Month of Onset
(for the Period, 1920-45, Inclusive)

	Months											
	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Cases	1	1	5	2	4	18	32	28	6	6	5	3

Reduction of numbers of human cases has paralleled reduction of numbers of animal anthrax cases. With an awareness of the mode of transmission of the infection to man, the proper disposal of the carcass of the anthrax animal and measures for control of the infection in exposed animals (quarantine and immunization), no human cases were observed in either of the last two epizootics: St. Bernard Parish in 1943 and the north central parishes in 1946. However, due to soil contamination with a long-lived type of spore-forming bacillus, occasional cases are still to be expected, as, for example, the 1945 case described herein.

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THE STATUS OF ABORTUS INFECTIONS IN THE UNITED STATES*

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Brucellosis in the United States presents a confusing problem to the medical profession. Because of its protean manifestations, the disease is not generally recognized. This failure in recognizing brucellosis is partly due to the fact that the majority of the cases are of the abortus variety, and observations indicate that *Brucella abortus* has been attenuated to such an extent that the disease has lost its clinical identity and an original diagnosis is difficult.

In consideration of the widespread prevalence of the organism in milk and dairy products and of the common usage of these foods in the United States, it is not surprising that the abortus variety should attain a more rapid and greater attenuation than would the suis or melitensis types. Extensive traffic in cattle and dairy products has assisted greatly in spreading the infection throughout the country, and this, also, has contributed to the attenuation of the organism, and to the difficulties presented in both clinical and laboratory diagnosis.

The true incidence of the disease is not known but judging from the possible sources of infection in cattle it is probable that brucellosis is most common. Evans[1] estimates that between 6 and 10 per cent of all cattle excrete the organism. Clark[2] states that 50 per cent of all milk-producing animals in the South are in herds infected with *Brucella abortus*, and Horn[3] showed that infection was present in 38.4 per cent of all dairy herds in the United States, which were estimated to include half of the dairy cows in the country. As early as 1911, Schroeder and Cotton[4] found that 14 per cent of all market milk contained *Brucella abortus*.

These figures indicate that the opinion of Gould and Huddleson[5] is probably correct in estimating that about 10 per cent of the population of the United States has become infected and that probably one per cent of these infected persons are ill with brucellosis at any given time. Wherever careful studies have been made, cases of brucellosis have been discovered, indicating that many of the cases are unrecognized and that the incidence of the chronic disease cannot be stated. The incidence of acute brucellosis has probably decreased because of the attenuation of the organism.

The distribution of the organism and the manner and frequency of contact with the organism are the chief factors that govern the change of pathogenesis of any infectious disease. In most such diseases contagion results from casual contact. Only in brucellosis, as noted in endemic areas, is there daily human ingestion of varying numbers of pathogenic organisms over a relatively long period of time. It must be admitted that either the patient has vaccinated himself against the disease or that he has it in some form, however mild or dormant.

The cow has played an important role in the attenuation of the organism. It is apparent that the disease has undergone the same changes in cattle as it has in the human. Veterinarians report that previously much time was devoted to attending cows that had aborted or delivered dead fetuses or that showed other definite signs of Bang's disease. In more recent years there has been a marked diminution in the incidence of these diagnostic signs. It is suggested that the disease is probably most prevalent in cattle and that the virulence of the infection has been altered by the cow.

A survey in a Southern rural section showed that 20 per cent of the cattle reacted positively for Bang's disease and in some dairy herds the incidence was as high as 60 per cent. It was consistently noted that only the finest stock were the positive reactors. These observations suggest that perhaps the better cattle were able to develop some immunity, whereas the poorer cattle were diseased without showing the presence of antibodies.

There is no other disease in which a host other than man plays such an important role. The widespread distribution of the organism in milk and the attenuation of it by the cow over a period of years has resulted in a chronic and obscure disease most difficult to diagnose and obstinate to treat.

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We may contend that the virulence and the antigenic properties of the organism are reduced to such an extent as to be unable to stimulate the formation of sufficient antibodies in the host completely to overcome the infection. This appears to be a plausible explanation of the reason for the existence of the disease in a chronic and obscure form and the absence of agglutinins in such cases.

This attenuation of *Brucella abortus* is not characteristic of the strain but rather may be attributed to the prevalence of the organism in cattle and the manner and frequency of contact by humans. The common use of contaminated milk is more conducive to the rapid attenuation of abortus as compared to suis or melitensis except in those areas where goat's milk is generally used. In Malta, brucellosis of melitensis origin exists as a low grade infection whose pathogenesis is similar to the abortus infections in the United States.

Thus it becomes apparent that the manifestations of brucellosis vary in different regions depending upon the extent and duration of the disease in animals. It is also apparent that the relative value of the laboratory procedures for diagnosis likewise varies depending upon the degree of attenuation of the organism in any particular area.

The widespread prevalence of the abortus infection among cattle for more than 150 years in the United States constitutes sufficient proof that the disease must have been most common in man prior to its recognition as a clinical entity. The prevalence and duration of the infection in endemic areas also suggest that the organism has been attenuated and in many instances, it exists as a non-pathogenic organism.

Though not generally recognized, this change of the pathogenesis of *Brucella abortus* has conformed with the basic principles of immunology and offers a satisfactory explanation for many of the difficulties encountered in both diagnosis and treatment.

Other diseases have undergone similar changes as a result of the attenuation of the organism and subsequent alteration of the manifestations has made clinical diagnosis most difficult.

The present situation for brucellosis closely resembles that for syphilis. In the great epidemic at the end of the fifteenth century, syphilis existed as an acute infectious disease manifested by high fever and symptoms of such severity that early death frequently resulted. This epidemic had all the characteristics of a virulent plague. Evidence indicates that with the rapid syphilization of the world, the virulence of the disease was attenuated and that within fifty years syphilis assumed the chronicity as presented today. As in the case of brucellosis the prevalence of the disease was denied by physicians until Wassermann devised a specific test in 1905.

Further observations indicate that the pathogenesis of syphilis and brucellosis bear a resemblance as regards the presence of antibodies. Stokes[6] states: "while it has been shown that agglutinins and lysins for the *Spirochaeta pallida* are formed, the amounts are insignificant so far as inhibitive effect on the disease is concerned." An analogous condition exists in endemic areas where brucellosis and Bang's disease are prevalent. Consequently the diagnosis of chronic brucellosis is as difficult as would be the diagnosis of syphilis without the aid of the Wassermann reaction.

The two diseases have other likenesses in common. The protean manifestations of brucellosis and syphilis are similar in that they have lost their clinical identity. They no longer present the symptoms of acute reaction following initial infection. They have lost their particular affinity for certain tissues, a characteristic yet retained by most other infectious diseases. Consequently the resultant pathology has been found in practically all of the tissues of the body. Both diseases may be congenital. In consideration of the clinical evolution of diseases, it becomes apparent that brucellosis and syphilis have undergone many similar changes.

This change in the pathogenesis of *Brucella abortus* is directly responsible for the difficulties encountered in both clinical and laboratory diagnosis. With the development of this chronicity, the symptom complex of brucellosis varies to such an extent that recognition is practically impossible. If one is familiar with the situation and knows that brucellosis presents such varying manifestations, the disease might be suspected and ultimately detected by means of differential diagnosis and the elimination of other possible conditions.

The classical description of brucellosis in old text books was based on observations of the acute infection. In endemic areas acute brucellosis is rarely seen and is usually of short duration.

This attenuation of *Brucella abortus* has detracted much from the value of laboratory procedures for diagnosis. The agglutination test is of little value in the diagnosis of chronic brucellosis of the abortus variety. These cases are of such low grade infection that one can hardly expect to find the presence of antibodies. In endemic areas agglutinins in any titer, however low, are of significance in the presence of suggestive symptoms. A negative agglutination, moreover, never excludes the possibility of infection. Other factors detract from the value of the agglutination test in the diagnosis of chronic brucellosis, namely the lack of a standard antigen. A marked variation has been noted in the agglutinability of the same commercially prepared antigens. In my opinion, a more sensitive antigen is necessary in order to detect low grade Brucella infections.

The intradermal test alone is not sufficient for diagnosis. A positive reaction indicates prior contact with Brucella but it does not distinguish past from active infection. Skin sensitivity has been noted in many patients who have never had evidence of disease. In endemic areas of Bang's disease a high incidence of positive reactions is to be expected.

The opsonophagocytic index is of questionable value and the procedure is not a practical one to be employed in the general practice of medicine. The difficulties encountered in growing *Brucella abortus* sharply limit the use of culture work in diagnosis.

In consideration of the changes that have taken place in brucellosis of the abortus variety, it appears plausible to assume that the complement fixation test would be the procedure best adapted for diagnosis of the chronic infection. Larson and Sedgwick[7] in 1913 used this test to suggest that bacillus abortus was pathogenic for humans long before Evans noted the resemblance between *B. abortus* and melitensis. The little work that has been done in the United States on complement fixation reactions in brucellosis has not been sufficient definitely to establish its value.

CONCLUSION

As a brief resume we may conclude that abortus infections are widespread throughout the United States in both humans and animals and that the organism has been attenuated so that clinical and laboratory diagnosis are difficult. Wherever brucellosis is prevalent and under circumstances such as exist in the United States, one may expect a change in the pathogenesis of the organism. The close similarity of brucellosis and syphilis should tend to bring about a more complete understanding of the problems presented by brucellosis.

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THE TOXIC EFFECTS OF DIGITALIS*

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INTRODUCTION

Upon reviewing the literature concerning this subject, it was apparent that, although a great deal had been written, there had been no attempt to combine this knowledge into a usable and practical reference for the practicing physician. It was with this fact in mind that the present paper was written.

GENERAL

The first description of the toxic effects of digitalis was written by William Withering in his paper, "*An Account of the Foxglove and Some of its Medical Uses.*" This was a description of the toxic effects of massive single doses of digitalis. Today one seldom sees this particular type of toxic manifestation, but sees rather, the toxic effects of digitalis due to the cumulative action of smaller doses. Today a great deal is being written concerning the relative toxicity of the different crystallized glycoside compounds, but since very little of this material has been clinically accepted, the present paper will deal only with the toxic effects of the standard products now in general and accepted usage.

For some time after the discovery of its therapeutic effect, digitalis was considered a panacea for anything that might be wrong or apparently wrong with the heart. Today, however, it is an accepted fact that digitalis is clinically indicated in the following conditions:

1. *Congestive Heart Failure*: Congestive heart failure is the principal indication for the use of digitalis. It is equally applicable in either left or right sided failure, and is not contraindicated by an arrhythmia or valvular defect. It has its greatest efficiency in congestive heart failure of nontoxic origin and disappointing results occur in congestive heart failure of other etiology.

2. *Auricular Fibrillation*: Digitalis should be used in auricular fibrillation only if it is caused by, or is accompanied by, congestive heart failure.

3. *Auricular Flutter*: The use of digitalis is the same as in auricular fibrillation.

4. *Paroxysmal Tachycardia*: Digitalis should be used in paroxysmal tachycardia only when the paroxysmal tachycardia is supraventricular in origin. Even then, its use should be restricted to selected cases and should be used only as a last resort.

There is no indication for the use of digitalis in the following entities:

1. *Coronary Thrombosis*: If congestive heart failure is associated with this, one must use his clinical judgment as to whether or not digitalis should be given.

2. *Angina Pectoris*: Digitalis is of questionable value if the origin is associated with congestive heart failure. There is no indication for its use if the patient is not in congestive heart failure.

3. *Pneumonia*: There is absolutely no indication for digitalis in pneumonia.

4. *Thyrotoxicosis*: If the patient is not in congestive failure, there is no indication for the use of digitalis. If the patient is in congestive failure, digitalis may be used with great caution, and with only minimal success.

5. *Diphtheritic Myocarditis or Other Infectious Myocarditis*: The indications for digitalis are the same as in thyrotoxicosis.

6. *Arrhythmias*: The arrhythmia must be either caused by, or associated with congestive heart failure before digitalis can be expected to accomplish any results.

7. *Valvular Lesions*: In general, it may be said that one obtains much poorer results in luetic valvular lesions than with rheumatic valvular lesions.

In their discussion of the toxicity of digitalis, Goodman and Gilman repeatedly emphasized that one must realize the toxic effects of digitalis are in reality simply an over extension of the normal effects. Consequently in this paper, the toxic effects will be compared with the normal effects.

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*The opinions and assertions in this article are those of the writer and are not to be construed as official or reflecting the views of the Navy Department.

EFFECTS OF DIGITALIS ON THE CARDIOVASCULAR SYSTEM

Chamberlain has divided the toxic effects of digitalis into three levels of poisoning; therapeutic stage, vagus, or inhibitory stage, and muscular stage.

Slowing down of the previously fast pulse rate is the principal indication in the therapeutic stage. At this stage, digitalis is exerting its principal effect on the myocardium alone.

In the second stage, the vagus stage, the digitalis is exerting its principal effect on the vagus and on the specialized conduction system of the heart. The pulse rate is decreased below 40-50, and various odd arrhythmias are set up, such as idioventricular rhythms and different degrees of heart block. Passage into this stage indicates the complete cessation of digitalis administration.

In the muscular stage, the pulse rate is increased, again due to the increased muscular irritability. Multiple extrasystoles are set up, bigeminal rhythm is frequently found, and the patient may eventually be precipitated into ventricular fibrillation and death. In this stage digitalis is exerting extreme toxic effects both on the myocardium and on the special conduction of the heart, and on the vagus.

Keeping these generalizations in mind, I will now go into a more detailed discussion of the toxic effects of digitalis on the cardiovascular system.

EFFECT ON HEART MUSCLE

Increased force of the systolic contraction is the principal effect of therapeutic doses of digitalis. All other effects on the cardiac muscle are subject to this. The drug increases the systolic tension several fold, whether the muscle is contracting at optimal or at high diastolic tension. It acts directly on the individual myocardial strands to increase the force of the systolic contraction, and is independent of other cardiac or extracardiac factors. This increased force of the systolic contraction in turn results in the heart being able to take care of an increased venous return which results in an increased diastolic filling and lowers the venous pressure. There is a decreased length of the systolic period. Thus there results an increased mechanical efficiency of the heart and consequent increased output which is probably the principal factor needed in decompensation.

The mechanism of the early toxic effects of digitalis on the musculature is unknown. The effects are thought to be due to a heightened irritability of the musculature to the conduction stimulus. The early toxic effects produce the following signs. If the patient has a normal pulse, and is in congestive heart failure, one begins to see multiple regular extrasystoles, or premature ventricular contractions. These extrasystoles arise in response to supraventricular impulses. If the patient is fibrillating, of course, extrasystoles are not noticed. With an increase in the toxic effects, one begins to see a pulsus bigeminus which is definite evidence of increasing intoxication, and is definitely dangerous. This arrhythmia has its origin in the ventricles and may progress to paroxysmal ventricular tachycardia and thence to ventricular fibrillation. Persistence of this arrhythmia indicates either lowering of digitalis dosage or complete discontinuance of its administration. Robinson states that one gets pulsus bigeminus only when 80 per cent of the minimal lethal dose has been given.

In 1943 Dearing, Barnes, and Essex carried out some experiments on cats to show the histopathologic effects of toxic doses of digitalis. None of the hearts of the cats showed structural change on administration of 40-50 per cent minimal lethal dose; one-fourth showed changes with 60 per cent minimal lethal dose; and seven-eights showed changes with 70-80 per cent minimal lethal dose. The following conclusions were reached: (1) With toxic doses of digitalis the changes that occur are; a degeneration of the myocardial fibers (vaculization, fragmentation, and disintegration in focal zones of myocardium), hemorrhage, and exudation of low grade inflammatory cells. (2) These changes occur most often in the papillary muscles, second in the left ventricular wall, next, in the intraventricular septum, and fourth in the right ventricular wall. They are usually located in the subendocardial regions. (3) These changes heal by fibrosis if the cat does not die. They become exceedingly difficult to locate within six to eight weeks after discontinuance of the digitalis.

EFFECT ON THE HEART RATE

Digitalis usually decreases the heart rate. This is probably accomplished by reflex stimulation of the vagus due to restoration of cardiac compensation. There is no discernible effect on the pacemaker except in toxic doses. In therapeutic dosages, there is no clinical effect of digitalis on the conduction system. Pharmacologically speaking, it is thought that digitalis begins depression of the conduction system even in therapeutic dosages. However, this depression is not manifest until toxic levels are reached.

When toxic levels are reached, the digitalis begins to exert an effect on both the conduction system and the exogenous nerve supply. Thus it is seen that digitalis may affect the heart rate by: effect on the myocardium (direct); effect on the conduction system, direct and by reflex; or by effect on the exogenous nerve supply, (principally vagus), direct and by reflex.

Extrasystoles (premature contractions), as previously stated are probably due to the hyperirritability of the ventricular myocardium. This is the most frequent type of arryhthmia and is not a contraindication to continued administration of digitalis. The premature contractions are frequently auricular, but this does not change the prognosis. This arrhythmia is one of the earliest signs of toxicity and usually begins with an isolated extrasystole. As toxicity increases the extrasystoles become multiple and regular. Further toxicity changes them to multiple irregular extrasystoles. Zwillinger states that an intracardial injection of magnesium sulfate (10 c.c. of 15 per cent solution) will stop extrasystoles of either auricular or ventricular origin. This procedure may be advantageous in cases where cessation of digitalis is undesirable. Magnesium sulfate may also be used in paroxysmal tachycardia, but fails to stop auricular flutter or fibrillation.

Sinus arrhythmia, in the absence of associated arrhythmias, is one of the earliest of the arrhythmias noticed. It is due to the stimulation of the vagus nerve. Sinus arrhythmia may be abolished by the administration of atropine. Its development is not an indication for the discontinuance of digitalis.

Auricular fibrillation is thought to be due to the direct action of digitalis on the conduction tissue. The exact mechanism of its initiation is not known. It may be reflex in origin, a too rapid restoration of mechanical efficiency. Auricular fibrillation is usually preceded by the presence of regular auricular premature beats, and may appear with or without other signs of toxicity. Opinion is varied as to whether the onset of this arrhythmia indicates the discontinuance of the drug. Actually, it depends on the relative insufficiency of the patient at that time, and on whether or not the physician thinks the patient is able to compensate fully enough to allow the withdrawal of digitalis. The fibrillation will usually disappear on the withdrawal of the drug. If precipitation into auricular fibrillation is associated with other signs of toxicity, discontinuance of digitalis is indicated. Schwartz warns that one must be particularly careful in administration of digitalis to children with an active rheumatic carditis.

Pulsus bigeminus is a comparatively early sign of toxicity. It occurs with increased frequency in patients with auricular fibrillation (either originating in congestive heart failure or from digitalis), patients under 40 years of age, and patients with rheumatic heart disease. Most patients receiving digitalis and with auricular fibrillation have premature ventricular systoles at the same time, though this arrhythmia is masked clinically. When the extrasystoles are forceful enough to open up the semilunar valves and occur regularly one after each normal systole, one obtains this pulsus bigeminus.

In approximately 40 per cent of the

cases 4 mg. of atropine subcutaneously will either abolish the arrhythmia or decrease it. The action of atropine in this instance is on the vagus, but the reason for the disappearance of the arrhythmia has not been clearly shown. If the arrhythmia does not respond to atropine, discontinuance or a decreased dosage of digitalis is indicated. Duration of the arrhythmia after withdrawal of the drug varies and is in direct relation with the severity of the heart disease. The presence of pulsus bigeminus decreases the prognosis but probably only because it appears in patients with more severe heart disease.

There are arrhythmias associated with delayed AV conduction which are due to digitalis in toxic doses. The digitalis decreases AV conduction by two methods: (1) vagal and (2) direct action—increased contraction results in an increased refractory period which in turn results in decreased conduction of the initiated auricular impulse. Depression of conduction through the node or bundle varies with the degree of digitalis intoxication. It may be divided into three stages:

1. Delayed conduction: In this stage, every impulse gets through but the intervals between auricular and ventricular systoles are lengthened. This is evidenced by an increased PR interval in the electrocardiogram.
2. Partial heart block: In this stage, impulses from time to time fail to reach the ventricle and a beat is missed. Auricular beats are regular and ventricular beats are missed, irregularly at first. Soon the dropping of beats becomes regular or remains irregular, so an auriculo-ventricular rhythm becomes established in which the two chambers beat in the ratio of 2:1, 3:1, or 4:1.
3. Complete heart block: When the AV node or bundle offers an absolute barrier to the passage of the impulse, the dissociation of the rhythms of the two ventricles is complete. The auricle beats at its own rate of about 70 per minute, and the ventricle at its inherent rate of about 35. This is the idioventricular rhythm, and is apparently due to a change in the location of the pacemaker.

This AV dissociation frequently leads to ventricular fibrillation which is apparently the cause of death in over-digitalized patients.

Heart block is not a prominent feature of toxic action of digitalis even though impairment of AV conduction usually appears at the dosage associated with therapeutic effects. Reid, in his paper, "The Bogy of Heart Block in Digitalis Therapy," states that there are no records of patients dying solely as a result of digitalis induced heart block. Quite a lot has been written concerning the point of AV delay at which digitalis should be stopped or at least decreased in dosage. Most authors used to believe that when the ventricular rate drops to 60 beats per minute, digitalis should be stopped. But, in view of the statement that heart block does not apparently result in death of the patient, it would seem that heart block is not a contraindication to continued digitalis therapy. Since most of the patients with heart block are also in severe heart failure, it is evident that if digitalis is stopped simply for heart block, quite a few will die from congestive heart failure.

Schwartz, in a review of this problem, states that digitalis in therapeutic doses may be of great benefit to patients with complete block and heart failure. The improvement in these patients takes place through direct action of digitalis on the heart muscle and not through an acceleration of the automatic beating ventricles. The ventricular rate is increased appreciably only after administration of digitalis in doses larger than those commonly employed in therapeutics. An increase in ventricular rate in complete block during administration of digitalis is a toxic manifestation, and, because of its undesirable effects, calls for its immediate withdrawal. Since it is difficult to appreciate the various types of toxic rhythms that manifest themselves in complete heart block during digitalis, it is advisable to prescribe the drug to such patients under careful graphic control. Groedel warns that any exercise in patients with a AV delay resulting from digitalis may convert the delay to heart block.

Auricular tachycardia is an arrhythmia that is not usually demonstrable clinically. No statistics have been published on its incidence, but many authors believe that it frequently precedes auricular fibrillation. This arrhythmia has its origin in an ectopic focus in the auricular muscle. Infrequently it may have its origin in the SA node. Auricular tachycardia is not a contraindication to continuance of digitalis therapy.

Ventricular tachycardia is an arrhythmia that has its origin in an ectopic focus in the ventricular muscle. Infrequently it may have its origin in the AV node or AV bundle. It may be either direct or reflex in origin. The occurrence of this arrhythmia is another reason why digitalis therapy involving any rhythmic changes should be closely followed by a graphic method. Electrocardiographic evidence of this arrhythmia, particularly if the QRS complex is changed in direction, should indicate immediate cessation of the drug since this arrhythmia frequently leads to ventricular fibrillation.

Auricular standstill is an arrhythmia that results from a severe SA block. Probably it is a result of the direct action of digitalis in large toxic doses. This arrhythmia is an indication for at least a decrease in the digitalis dosage. If the standstill is not broken by decreasing the dosage, the digitalis should be stopped. The prognosis is definitely decreased by the appearance of this arrhythmia, probably because it is associated only with a severe form of toxicity.

Paroxysmal tachycardia of auricular origin is a comparatively infrequent arrhythmia believed to consist of a series of rapidly recurring extrasystoles which completely submerge the physiologic rhythm. The electrocardiogram shows complete complexes, occurring at rates of 120-250 per minute. This paroxysmal tachycardia occurs most frequently after middle life, and occurs at a low toxic level of digitalis. Frequently there are not associated toxic symptoms. Continuation of digitalis is not particularly contraindicated.

Paroxysmal tachycardia of venticular origin may have its origin in the AV node, stem of the bundle, or in the ventricle itself. When the impulses arise in the AV node or supraventricular part of the bundle, the PR intervals are shortened. Inversion of the P waves is common. Also the contraction of the ventricles may be simultaneous with that of the auricles, and the P wave will be buried in the QRS complex. Many variations of the electrocardiogram are seen in this arrhythmia. There is an increased incidence of this arrhythmia in the presence of auricular fibrillation. Recently several cases have been reported in which paroxysms consist of regular alternation of upward and downward ventricular complexes, probably due to ectopic beats arising alternately in the right and left ventricular muscle.

In general it may be stated that any increase in the heart rate without obvious physiologic cause in a patient who is receiving digitalis, or any apparent failure of digitalis to slow the fibrillating heart should be regarded with suspicion. In proven cases of paroxysmal ventricular tachycardia, digitalis should be stopped immediately due to the imminent danger of precipitating the heart into ventricular fibrillation.

Ventricular fibrillation is the end point of digitalis toxicity, since it is stated that most over-digitalized patients who die do so as a result of this arrhythmia. Every care must be taken to avoid this arrhythmia. It is usually preceded by other types of toxic rhythms as indicated above, during which digitalis should be stopped or the dosage decreased. Schwartz and Jezer state that there are some patients who, while in congestive heart failure, seem predisposed to the development of this arrhythmia. These patients are usually over 60 years old, have had clinical evidence of heart disease for 10 years, and have had at least two episodes of congestive heart failure. When this arrhythmia occurs immediate cessation of digitalis is indicated. One method suggested for resuscitation of the fibrillating heart is the injection of 0.5 per cent solution of potassium chloride into the carotid arteries so that some gets into the coronary artery. One or two minutes later a .023 per cent solution of calcium chloride is introduced by the same route to restore the heart to normal rhythm.

Other arrhythmias have been reported, but those not here discussed are comparatively rare. Examples are pulsus alternans, phasic irregularity.

EFFECTS OF DIGITALIS DEPENDENT ON RESTORATION OF CARDIAC EFFICIENCY

The normal effect of digitalis on the cardiac output is determined by the heart rate, the heart size, the stroke volume, velocity of the blood flow, peripheral resistance, and venous return. In normal hearts, the venous return is adequate, the peripheral resistance is approximately normal and the heart rate is comparatively unaffected by digitalis. Thus the heart is at maximum efficiency and is maintaining normal output. However, due to digitalis, the capacity of the ventricles is reduced due to the direct cardiotonic action. This in turn results in a decreased stroke volume and decreased cardiac output. The decompensated heart is usually dilated and mechanically inefficient. Due to the direct cardiotonic action, the heart is returned to the normal maximum efficiency. Stroke volume is increased and the normal cardiac output is attained.

In passing, one may say that the good effects of digitalis may be offset if any toxic arrhythmia is induced. In short, restoration of maximum cardiac efficiency is possible only with therapeutic drug levels. The normal effect of digitalis on the venous pressure, in general, is that the pressure is reduced due to the increased cardiac output. As explained above, any toxic arrhythmia interfering with the cardiac output may result in an increased venous pressure.

Congestive heart failure increases the circulatory volume. Digitalis, by restoring the normal cardiac output, decreases the circulatory volume. These findings correlate with the recent reports in which McGuire, Johnson and Calvus stated that in patients with congestive heart failure in whom 70-80 per cent of the calculated minimal lethal dose of digitalis was accidently administered with the resulting toxic arrhythmias, the congestive heart failure was not alleviated, but in some cases actually increased.

EFFECT OF DIGITALIS ON BLOOD PRESSURE

Comparatively little work has been done on this point. Effects on the normal blood pressure are highly variable and so far, no toxic effects have been recorded. In general the following effects may be expected: no significant change in a normal person; in patients in congestive heart failure, an increase if originally low, and a decrease if originally high.

EFFECT OF DIGITALIS ON CORONARY CIRCULATION

Literature is very controversial concerning this subject. However, it is generally believed that digitalis in therapeutic doses has no effect on the coronaries. This belief is fairly well substantiated by the recent work of Dearing, Barnes and Essex who, after experimenting with approximately 20 cats, state that the calculated therapeutic doses produced no appreciable change in the coronary flow.

Further work by Dearing, Barnes, and Essex revealed that calculated toxic doses of digitalis resulted, a definitely decreased coronary flow which occurred from four to six hours after administration of the drug, and which persisted for several days after discontinuance of the drug. There was no correlation between the decreased coronary flow and changes in the pulse rate or blood pressure. Fenn and Gilbert state that digitalis is capable of producing anginal pain by constriction of the coronary arteries, but this does not happen in every case, nor can it be predicted in any case. In conclusion, it may be said that there is indication that coronary flow is reduced by over-digitalization, but that until further comfirmatory work is done, no definite statement can be made.

ELECTROCARDIOGRAPHIC EFFECTS

Changes in the T wave are shown early on the electrocardiogram of a digitalized patient. The amplitude is decreased, flattened, and finally inverted. T_1 is inverted and T_3 retroverted, or the opposite. Dearing estimates that a calculated 20-30 per cent minimal lethal dose (which is a therapeutic level) will bring about changes in the RST segment. There is usually a depression of the RST segment which is due to direct action on the muscle and is not abolished by atropine. Later the prolongation of the PR interval occurs. It may be either vagal or direct in origin. There is also a shortening of the QT interval due to decreased duration of ventricular systole. It must be emphasized that these changes are frequently absent and can not be used as a quantitative estimation of the digitalis level. There are unusual electrocardiographic changes present due to the toxic arrhythmias which will not be described here.

EFFECTS OF DIGITALIS ON THE GASTRO-INTESTINAL TRACT

Oral digitalis is adequately absorbed by the intestinal tract under nearly all circumstances, including chronic passive congestion of the enteric mucosa due to congestive heart failure. Local irritation is apparent even when the drug is taken with meals to avoid this action. The drug is adequately absorbed within two hours, and exerts full action on the heart within six hours. Digitalis in therapeutic doses exerts no abnormal toxic effects.

The toxic manifestations of digitalis on the gastrointestinal tract are: anorexia, nausea, vomiting, diarrhea, increased salivation, and abdominal discomfort and pain. All of these manifestations are apparently reflex in origin, with the possible exception of diarrhea. They occur equally as much with intravenous administration as with oral administration. Experiments with gastrectomized animals reveal that the same toxic symptoms occur.

Anorexia is probably the earliest sign of approaching tolerance in the administration of digitalis. It occurs one to two days before nausea and vomiting (that is, if the dosage is increased), and persists until the digitalis is reduced in dosage, or at times, until it is stopped completely. Anorexia is not an indication for discontinuance of digitalis, but is an indication that tolerance is being approached and that dosage can not be increased much further without the appearance of other toxic symptoms.

Nausea and vomiting may be considered together as they are apparently due to the same mechanism. Nausea is an early indication of toxicity and tends to appear in recurring waves. Vomiting occurs after the onset of nausea and is variable in duration and in amount. Both symptoms have been proved not to be due to local irritation of the gastrointestinal tract. Rather it has been shown that they are due to the stimulation of a medullary emetic center. These stimuli apparently come directly from the over-digitalized heart and are carried mainly by the sympathetics, and possibly the vagus.

Repeated doses of digitalis may depress the vomiting reflex while simultaneously increasing the intensity of the cardiac poisoning so that after an initial period of vomiting, the continued administration of these drugs may fail to produce emesis, and even cause death without further vomiting. These experimental results suggest the need of caution in relying on nausea and vomiting as measures of the degree of cardiac poisoning in the clinical use of digitalis. The most probable explanation of the change in response of the vomiting reflex appears to be that doses of digitalis may continue to increase the cardiac poisoning while at the same time directly depressing the vomiting reflex.

Very little work has been done on the subject of increased salivation as a phase of digitalis poisoning. It is a comparatively early sign of toxicity and is apparently due to a sympathetic stimulus set up by the over-digitalized heart. Its clinical significance cannot be evaluated at the present.

Diarrhea is a comparatively infrequent manifestation of digitalis toxicity. It has been suggested that the diarrhea is due to an increased emptying time of the stomach as a result of the local irritation of the gastric mucosa. It has also been suggested that the diarrhea may be the result of the changing fluid balance which occurs when the heart is recompensating and congestive failure is being done away with. Diarrhea may occur at any level of toxicity and is not an indication of any change in digitalis dosage.

Abdominal discomfort and pain are manifestations that also are fairly rare. At the present, no explanation of their mechanism is available and their clinical significance can not be properly evaluated.

CEREBRAL EFFECTS OF DIGITALIS

The effects of digitalis on the central nervous system may be divided into three main parts; the reflex stimulation of the medullary vomiting center as explained previously; the general effects on the central nervous system; and the so-called digitalis delirium.

The general effects on the central nervous system are present even in patients who have received a calculated therapeutic dosage of digitalis, but perhaps are intensified with toxic dosages. Many effects have been listed. Some are headache, fatigue, malaise, drowsiness, excitement, restlessness, increased iritability, nervousness, inability to think, and languor. None of these symptoms are an indication for discontinuance of digitalis unless they reach such a point that the physician thinks they are interfering with the convalescence of the patient.

The mechanism causing the general cerebral effects is not clearly understood. The headache is general in distribution, is not particularly severe and seems to increase as the patient becomes recompensated. It has been suggested that not only the headache, but the other symptoms as well may be due to the changing fluid balance which results from recompensation of the heart. It has also been suggested that the headache is due to a reflex cerebral vasoconstriction caused by the varied increase in cardiac output of the digitalized heart, or perhaps caused by actual sympathetic impulses originating in the decompensating heart.

The question of digitalis delirium is even more clouded. Probably most physicians follow the teachings of Weiss, who states—"In the psychosis which occurs after the administration of digitalis, we are dealing with a cerebral circulation which is *a priori* abnormal due to chronic sclerotic vascular changes or to circulatory changes. If sudden general circulatory changes are produced through digitalis or rest, a psychosis develops because of sudden changes in the cerebral circulation and because of the physicochemical change associated with the removal of large amounts of edema. Whether the entrance of toxic chemical decomposition products of the body or other chemical changes are responsible for the psychosis we are unable to say at the present." This is certainly a very tenable explanation, since it has been shown that in 97 per cent of the reported cases the patient has been over 50 years of age and has had advanced generalized arteriosclerosis.

Again, however, we are brought face to face with the new evidence accumulated by Dearing in his experiments with the histopathologic changes of digitalis toxicity in cats. Dearing found that:

1. Thirty per cent of calculated minimal lethal dose (the therapeutic dosage) either in rapid digitalization, or maintenance dosage did not result in any tissue changes.

2. Sixty per cent of the calculated minimal lethal dose was the smallest dosage to give tissue changes. The changes increased with increased dosage and did not occur until after six or seven doses.

3. Changes affected the large and small pyramidal cells of the cerebral cortex and consisted of the following: swelling of the cell body, vacuolization of the cytoplasm, varying grades of cytoplasmic and nuclear degeneration, complete liquefaction of the cells, pyknosis of the cells, complete cell degeneration with following satellosis. This reaction varied from focal to diffuse in location. If the animal recovered, the lesions disappeared in three to four weeks.

So if, as Dearing suggests, the actions of digitalis in the cat are analogous with its action in the human, there is a much firmer basis on which to explain the cerebral symptoms. One must also remember the older explanation of change in cerebral fluid balances.

In digitalis delirium the following symptoms may be seen: disorientation, particularly of time and place; confusion; actual delirium, progressing to stupor; illusions and hallucinations; temporary aphasia (apparently motor in type); temporary impairment of memory; and rarely convulsions. Carr states that to reach a diagnosis of toxic digitalis delirium, one must satisfy the following standards: (1) absence of other causes of delirium; (2) presence of other toxic symptoms; (3) onset of delirium as decompensation recedes; (4) previous administration of a large amount of the drug; (5) rapid clearing of the symptoms when digitalis is withdrawn, and (6) reappearence of the same symptoms when digitalis is readministered. In the event of diagnosed digitalis delirium the following steps are recommended by Smith as the proper treatment. After recognition, discontinuance of digitalis, then an increased fluid intake and restraint. Under this regime, Smith states that 90 per cent of the patients will recover within four to six days.

EFFECTS ON VISION

Many effects on vision have been noted and recorded as being due to toxic doses of digitalis. Some of these are halo vision, color vision, diplopia, bizarre varieties of light sensation, inability to focus eyes, "angle" vision, oscillation of objects, temporary blindness. Objectively, little has been found. Ophthalmoscopic examination has been repeatedly negative. Various other findings have been reported in isolated cases, mild conjunctivitis with peripheral corneal injection, and uneven dilatation of pupils. Exophthalmos and even nystagmus have been reported.

There is no constancy in the time of the appearance of these symptoms. The milder symptoms usually appear early in the digitalis course. The milder symptoms most frequently manifest are the bizarre varieties of light sensation and scotoma. Among these varieties are flickering, flashing, blurring of vision, amblyopia, and all types of spots and sparks. Diplopia and inability to focus the eyes are probably the next most common symptoms. Halo vision and color vision are relatively infrequent. Color vision has been reported as being white, green, lavender, red, blue and most frequently yellow. It may be partial, in spots, general, transitional, or of variable intensity. It must be remembered that color vision may be due to other factors; that is, yellow vision may be due to cannabis indica, amyl nitrite, and picric acid poisoning.

The other visional aberrations are comparatively rare and of no practical clinical significance. Unless the above symptoms are accompanied by other signs of toxicity there is no indication for cessation of digitalis administration.

Several mechanisms have been suggested as the causal agent for these symptoms. Jackson has suggested that the color vision is due to action of digitalis on the tensor choroidae and dilatation of the vessels of the iris. Actually, it has been found that the vessels of the iris are dilated more than usual during these visual episodes. Weiss has suggested that colored vision and perhaps the other visual manifestations may be the result of spreading of excitation waves from the vagus center to the visual centers of the mid and hind brain.

EOSINOPHILIA

This is a relatively rare manifestation of digitalis toxicity. Few substantiated cases have been reported, and the eosinophilia has varied from 6 to 30 per cent. In order to be classified as a digitalis eosinophilia, the eosinophilia must not be pre-existent of the administration of the digitalis; there must be a noticeable increase within 12-18 hours after administration of the drug; there should be a decrease in eosinophilia within two days after cessation of the digitalis. There is some evidence that this eosinophilia is the result of vagus stimulation by digitalis. It is fairly well clinically accepted that direct vagus stimulation may result in an eosinophilia which is abolished by atropine, and increased by pilocarpine. Since, writers have reported that the digitalis induced eosinophilia has been abolished by atropine. This phenomenon has no proved clinical significance and its appearance does not affect the digitalis dosage used.

ALLERGIC EFFECTS

This is one of the more rare manifestations of digitalis administration. It perhaps should not be considered as a true toxic manifestation since it occurs even on initial dosage of digitalis and is due neither to an initial massive dose, nor to a cumulative effect of digitalis. A typical allergic picture evolves. The patient has fever, general pruritis, urticaria, joint involvement, edema of the face, and rarely an eczematoid reaction. There is no change in pulse rate or blood pressure. The skin test is negative as with all other drug allergies. Digitalis allergy is diagnosed on the history, objective findings and results of administration and withdrawal.

MISCELLANEOUS EFFECTS

General muscular weakness is an effect of digitalis toxicity that has been reported several times. It is rather an unusual manifestation and shows some variation in the time of its appearance. It has no particular clinical significance and its appearance does not indicate cessation of the administration of digitalis. Neurologic examination is normal in every respect and muscle strength returns in approximately four days after discontinuance of the drug.

There have been various case reports in which embolism has been blamed on the administration of digitalis. The mechanism of this is probably due to the transition of the arrhythmia from auricular fibrillation to less toxic rhythms. This results in the shaking loose of any mural thrombus that has been formed. Fortunately comparatively few cases have been reported in which the embolism can be directly traced as a result of administration of digitalis. This phenomenon results from the administration of therapeutic doses of digitalis and there is no particular clinical indication that it is going to happen.

As previously stated, digitalis has comparatively disappointing effects in the congestive heart failure with hyperthyroidism as an etiology. In regard to the accentuation or decentuation of toxic effects of digitalis in these states, it has been more or less proved that there is no significant difference in the response.

Many authors have warned that calcium should never be administered during digitalis therapy due to its synergism with digitalis. However, Smith and Hoft after a series of conclusive experiments have stated that the lethal effects of calcium and digitalis are neither synergistic nor even completely additive. The only danger of injection of calcium with digitalis is its early inherent toxic action on the heart muscle.

Many cases have been reported in which digitalis was administered to patients with congestive heart failure and edema. No effect was noticed until the edema began to subside and then toxic effects seem to be precipitated all at once. This effect has been proved to be due to the fact that digitalis has a tendency to accumulate in the edema fluid and when the edema subsides, the digitalis is released in toxic doses.

SUGGESTIONS FOR AVOIDANCE OF TOXIC EFFECTS

The first point that one must consider is whether or not digitalis is actually indicated in the case. The indications for administration have been listed above. Actually, the contraindications for digitalis therapy is the lack of indication. Ashburn has outlined the following points as the common misusages of digitalis: to slow heart in simple tachycardia of nervous origin (effort syndrone or goiter), or toxic origin (diphtheria, rheumatic fever, pneumonia); paroxysmal tachycardia; valvular and other abnormalities, with possible exception of mitral stenosis, unless there is myocardial insufficiency; quantity *per se* of the dose (too small a dosage, too large a dosage).

The following list may be used generally as indication for the discontinuance of digitalis, or at least its reduction. Of course it must be tempered by the judgment of the physician. (1) Severe and increasing nausea and vomiting; (2) any change in regular rhythm to an arrhythmia, particularly sudden slowing of the cardiac rate, slowing of the cardiac rate to 60 or below, any evidence of an increasingly delayed AV conduction, or changing of extrasystoles to pulsus bigeminus; (3) headaches, dizziness or disturbed vision, together with other toxic symptoms.

In closing, it should be said that with the appearance of so many new digitalis drugs and derivatives on the market today, it becomes increasingly necessary for the physician to select one dependable preparation and to limit his experience to this one preparation. He should make every effort to understand this preparation thoroughly; its variability, its limitations, its eccentricities and most important of all its therapeutic and toxic ranges.

SUMMARY AND CONCLUSIONS

In this paper, the various toxic manifestations of digitalis have been presented—as they affect the cardiovascular system, the gastrointestinal system, the central nervous system, and other bizarre manifestations. An attempt has been made to explain the mechanisms behind these different toxic effects so that the practitioner may understand the physiologic basis of these effects instead of learning them by memory. In view of the world wide use of digitalis, it is apparent that the up to date physician must know the limitation of this extremely valuable but relatively dangerous drug. It is hoped that this paper will help to give the doctor a more thorough understanding of the action of digitalis and in particular the danger signs of its toxicity.

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INFECTION OF THE MOUTH AND PHARYNX

A REPORT OF 98 CASES

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A discussion of certain infections with resulting abscess formation in the soft tissues that make up the walls of the mouth and pharynx should be of interest to the general practitioner, the internist, as well as the head and neck specialist, for any of these may be summoned to see such a patient. As long as there exists infected tonsils, infected teeth, and infected ears, from time to time there will develop extensions of these infections into the deeper structures of the neck. This involvement always occurs as a secondary affection, yet it is of primary importance.

Ther are four types of abscess formation to be considered. They are peritonsillar, perialveolar, parapharyngeal, and lastly, retropharyngeal abscess. The perialveolar abscess may progress into Ludwig's angina if in the lower jaw or develop into a palatal or buccal phlegmon if in the upper jaw.

The fascial planes of the neck serve as distinct natural barriers when infection does develop. It is of paramount importance to keep this in mind because too eager or ill-advised surgical interference increases the rapidity of the extension and therefore delays localization or resolution. The word "space" has come into use in referring to certain areas that become sites of abscess formation. "Spaces", as is known, do not exist as such in the neck, but rather the abscess itself creates this space by destroying the soft areolar tissue and pushing aside the muscles and ensheathed blood vessels and nerves. Almost any site might conceivably become infected, but experience has shown that certain sites are more susceptible.

A review of the records of patients in the Oak Ridge (Tennessee) Hospital and Clinic[*] for a two-year period from December 1, 1943, to December 1, 1945, revealed a series of case histories of 98 such patients who fell into this category. There were 66 cases of peritonsillar abscess, 26 with perialveolar abscess, five with parapharyngeal abscess, and one with retropharyngeal abscess. It became apparent from a review of this series that while most of these patients were acutely ill upon admission, almost all responded rapidly to treatment. Certainly one did not observe any of the complications which have been known to develop when abscess exists in these locations. From peritonsillar abscess, a parapharyngeal abscess may develop. There may occur a thrombophlebitis of a neighboring vein, and subsequently a fatal septicemia may ensue as a result of a parapharyngeal abscess. Cases of fatal hemorrhage have been reported that were due to an erosion of an artery. One patient who was admitted to the hospital with a subacute mastoiditis was found upon examination to have had a complete loss of his upper palate on one side. This had resulted from an infection arising from an abscessed upper molar tooth which progressed into a phlegmon and osteomyelitis of the entire upper jaw of that side. Unfortunately it had occurred before the advent of sulfonamides. Retropharyngeal abscess has been known to extend downward and cause a diffuse mediastinitis; it may cause such severe dyspnea by edema of the glottis that a tracheotomy is necessary, or it may rupture spontaneously and fill the respiratory tract to suffocate the patient.

PERITONSILLAR ABSCESS—SIXTY-SIX CASES

This type of abscess, commonly known

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as "quinsy" is usually an excruciatingly painful condition, and fortunately is the most easily managed of the four types of abscess formation. In this group there were two patients who were 8 years of age and there were four over 50. An attack of acute tonsillitis preceded the onset of the abscess. In two patients, tonsillectomy had been done many years earlier but some tonsil tissue still existed. One of these had but a small but painful abscess formation which gave relief when incised. The second, who likewise gave a history of a tonsillectomy some years earlier, stated that the physician had had some operative difficulty with the presently involved side. Upon close inspection, a portion of the upper pole of the tonsil could be seen bound down by dense scar tissue and overlying this was a very red and tense entire soft palate and peritonsillar area. Even though a generous incision was made, purulent exudate did not exude until a curved hemostate was inserted deeply and then opened. In 53 of the patients, the peritonsillar abscess was incised at the initial visit; however, 22 of these patients had such severe systemic symptoms of marked pharyngeal involvement that it was felt hospitalization was indicated.

Treatment consisted of hot saline throat irrigations (not a gargle) given every one or two hours along with which one of the sulfonamides or penicillin was administered. In 18 of the 22 hospitalized patients, it was deemed advisable to respread the primary incision to permit drainage of accumulated exudate. Recovery occurred in from three to five days. One patient, in spite of hospitalization for a severe follicular tonsillitis and treatment with throat irrigations and sulfonamides, developed a bilateral peritonsillar abscess. When the patient was seen originally in consultation on the eighth day, the tonsils appeared very large and the peritonsillar tissue and soft palate appeared very swollen. It was not realized until the following day that this was a bilateral peritonsillar abscess which existed no doubt before and had not been recognized as such. Incision on each side gave relief and confirmed the tardy diagnosis.

It was customary to request all patients who had the peritonsillar abscess opened in the clinic to return the following day. This was so an opportunity might be afforded to respread the incision if it were necessary. In most instances this procedure did release an additional amount of exudate and permitted a more prompt recovery. No anesthesia, either topical or by infiltration, was used preceding the opening of the peritonsillar abscess. This applied also to the two 8 year old children. A cataract knife is used to make the incision at a point where a horizontal line through the free edge of the soft palate crosses a vertical line just mesial to the base of the anterior tonsillar pillar. Immediately following a long curved hemostat pointing mesially is inserted through the incision and then opened as it traverses the posterior pole of the tonsil. This method has proven more satisfactory than trying to select the point of greatest bulging for incision of the abscess.

PERIALVEOLAR ABSCESS—TWENTY-SIX CASES

As the name would imply, this infection is of dental origin. It may develop from an apical abscess of a tooth, or, it may arise from dental operation on these teeth or from infection pre-existing or developing at the site of fracture of the mandible. In this group there were 14 patients who exhibited sufficient inflammation and edema about the anterior pillar of the tonsil and the lateral side of the tongue to displace the tongue itself well over to the opposite side. Pain and swelling in the sublingual region, stiffness of the jaws to almost complete trismus, and external swelling under the angle of the mandible are the usual findings. It was felt that at least five of the 26 patients would have developed a deeper parapharyngeal involvement if penicillin had not been given. There were six patients who had the disease process develop in the upper molar area. The inflammation extended mesially to involve the soft palate and inferiorly to the inter-alveolar sulcus. Trismus was marked in these patients. Penicillin and intraoral irrigations of hot saline were used to treat these.

There were three cases which could be traced to fracture of the mandible, with concurrent apical dental infection at the site of fracture. One case was so acute that incision and drainage externally was carried out at the initial visit. The other two, who were given penicillin with an appreciable help, also had external drainage. One was by incision and the other developed an external phlegmon which later broke down in three different places.

The remaining 16 of this group were given either sulfonamides or penicillin, or both, in addition to hot saline intraoral irrigations. However, nine had to have the abscessed cavity incised intraorally while the remaining seven subsided without surgical intervention. One patient upon admission had such severe sublingual swelling, displaced tongue and trismus, and dehydration due to dysphagia that it was necessary to give intravenous dextrose and saline, in addition to penicillin. Yet recovery occurred in five days without surgical intervention.

In this same group of 26 patients, only two had had dental operations, that is, extractions. In other words, infection had pre-existed in the molar teeth and extractions were done for relief. One patient had had infection in both the first and second molars which were extracted. Trismus and marked swelling developed but recovery was prompt after sulfonamides were given. The second patient did not fare so well. Three days following an extraction for the one of three infected lower molars the x-rays of which showed adjacent bone absorption, a marked submandibular swelling developed in addition to swelling and induration on the buccal side of the alveolar process. The infection steadily progressed in spite of large doses of penicillin given at three hour intervals day and night for three weeks. For a while it was thought that a massive necrosis of that portion of the mandible was going to develop. Finally, however, signs of localization became evident. The submandibular swelling receded but the perialveolar and buccal swelling increased. By mandibular nerve root block, an adequate incision was made intraorally over this area which released a small amount of purulent exudate. This procedure was repeated three days later with more satisfactory results and again a week later with subsequent distinct improvement. Numerous small bone sequestra were given off at intervals from the tooth socket as well as through the incised area. The patient ultimately made a recovery although it was protracted.

PARAPHARYNGEAL ABSCESS—FIVE CASES

The usual site of entrance of infection to form a parapharyngeal abscess is just lateral to the tonsillar fossa. It may follow tonsillitis, peritonsillar abscess which is not properly drained, dental infection, dental operations, or tonsillectomy, usually when done under local anesthesia. A patient with a parapharyngeal abscess usually presents marked trismus, pain upon swallowing, and sometimes dyspnea. There is external swelling under the angle of the mandible and on the lateral side of the neck. Intraoral examination reveals a swelling lower and more lateral than the peritonsillar or a perialveolar type of infection. This type of abscess is more potentially serious than the two previous ones because if untreated or unrecognized, it may penetrate to involve the carotid sheath. By virtue of its deepness and being well covered, external fluctuation should never be waited for; it is a late sign. Once the diagnosis is established, it is important to promote drainage and the author feels this can be accomplished in most instances by the intrapharyngeal route. The method employed is to incise vertically the mucous membrane of the tonsillar fossa or, to incise between the tonsil, if present, and the posterior pillar. A long slender curved pointed hemostat is inserted through the incision to a depth of at least one inch and then spread. This method gave adequate drainage. No external incision was needed.

In this group of five patients, tonsillitis was the cause of one, an abscessed lower third molar the cause of the second, and the remaining three developed following exodontia. In fairness to the exodontists, two of the individuals had severely abscessed molar teeth as well as poor dental hygiene to increase the hazard of extraction by local anesthesia. The third had a severely infected wisdom tooth which was removed under nitrous oxide anesthesia so obviously it could not be ascribed to faulty novocaine injection. All five patients had elevated white blood cell counts, one being over 18,000, fever, marked trismus, and extreme dysphagia. Examination revealed definite inflammation and edema about the entire tonsillar fossa of the involved side. Curiously, the one who had had an attack of tonsillitis showed no especially infected tonsils when first seen. Two of the patients had such marked dysphagia that it was necessary to give glucose and saline solution intravenously to maintain the fluid balance. All five patients received penicillin every three hours which seemed to prove distinctly beneficial. Gentle mouth irrigations of hot saline solution were given at frequent intervals. Intrapharyngeal incision and drainage as described above was done on three of the patients. The abscess ruptured and drained spontaneously in the two patients who had had extractions with novocaine anesthesia. Once drainage was established, the patients made rapid progress. The penicillin was kept up for two more days, however, to hasten recovery. No further complications or sequelae occurred in this group.

RETROPHARYNGEAL ABSCESS—ONE CASE

An acute retropharyngeal abscess occurring in an adult when not secondary to cervical vertebra disease but rather due to infection in the ear and tonsil is unusual. The acute type is usually found in the child of 3 years or younger.

The patient with this affection was a white married female, 27 years of age and six months pregnant. The history brought out that she had had a chronic otitis media for some years and a recent attack of severe tonsillitis. Her chief symptoms were difficulty in swallowing which became progressively worse over a period of eight days, and a sensation of fullness in the back of her throat. Examination revealed a marked midline swelling protruding from the pharynx. She also had considerable apprehension. Her fever was just over 100, white blood cell count 12,600, and x-ray of the soft tissues of the neck confirmed the clinical diagnosis. The abscessed cavity was incised and suction used as it was done. Spot cannula suction into the cavity was used on each of three successive days. Penicillin was given during the entire time of hospitalization and the patient made an uneventful recovery within a week.

SUMMARY

A group of 98 patients with infections in the soft tissues of the neck arising from infection in the mouth and pharynx is discussed.

There were four main groups of abscess development which were individually considered.

Prompt recognition and drainage supported by either sulfonamide or penicillin therapy is important if serious complications are to be avoided.

SIGMUND FREUD*

GENE L. USDIN, M. D.
NEW ORLEANS

If we were to travel far to the north in the province of Moravia in what is now Czechoslovakia, we might pass through a small country town, Freiberg, typical of the Austrian village of the nineteenth century. Here in the peaceful pastoral village among the rolling foothills of the Carpathian mountains—half a mile from a dense forest—was born Sigmund Freud. Freud writes: "Meine Eltern waren Juden, auch ich bin Jude geblieben" (My parents were Jews, and I have remained a Jew).

Somewhat off the market place and close to fields and pastures, at 117 Schlossergasse, Jacob Freud lived comfortably with his family in a rather small, two-story house without ornamentation, constructed of plastered bricks, with a slate roof. In 1885, Jacob, then over forty years of age, married a second time. His bride, Amalia Nathansohn, was not yet twenty. The following year Sigmund was born.

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Sigmund's father's family had settled for a long time on the Rhine, at Cologne, but as a result of the persecution of the Jews during the fourteenth or fifteenth century, they fled eastward, and in the course of the nineteenth century, they migrated back from Lithuania, through Galicia, into German Austria.

At the time of Sigmund's birth Jacob Freud already was a grandfather. Emanuel, the eldest of the two sons of his first marriage—then in his twenties—had a son, John, one year old, and a newborn daughter, Pauline. Thus little Sigmund had a nephew, John, a year older than the uncle. As Freud's development was greatly nifluenced by the conflicts between the two lads, this peculiar relationship is well worth mentioning.

Like Goethe, who was one of his favorites, Freud came into the world black in color. But whereas in Goethe's case this was due to the cyanosis of impending suffocation, Freud's darkness was due to a covering of black down, so much so that his mother called him "a little blackamoor." Possibly some of his antagonists might find this birth symbolical of the part he was destined to play in the world, a part which they might term satanic. He himself does not seem wholly to deprecate such a charge seeing that the motto he has chosen for his book "Die Traumdeutung" is "Flectere si nequeo superos, Acheronta movebo" (If I cannot influence the Gods of Heaven, I will stir up Acheron). It is interesting to note that three weeks before his birth, a traveling gypsy had told Freud's mother that she was about to give birth to a great man. Subsequently in Vienna, an itinerant fortune-teller informed Sigmund's parents that the boy was destined to become a minister of State, an incredible prospect in the Austria of those days for any one who was not of noble birth. The most remarkable point, however, is that forty years later Freud dreamed of the childish trifle. Using Freud's own basic concepts of dream analysis, it is but a simple deduction to see that the lad's ambition was something quite out of the ordinary.

In Freud's story of his childhood, we learn a good deal about his nurse, Nanny, "that prehistoric old woman." She was very ugly but very clever and efficient, and she often treated him quite unpleasantly. She scolded him bitterly and spanked him when he did not show sufficient appreciation for her standards of cleanliness. Yet he gave her his love and all his pennies. She was sorely missed when she later disappeared—with more than the family's pennies.

We also get a vivid picture of the alternately friendly and hostile relations between the young uncle and his older and stronger nephew, John. They were inseparable. They loved each other and fought each other. Occasionally John was a tyrant who mistreated his younger playmate. But the little uncle had courage and knew how to stave off permanent submission. For years the family remembered the manner in which the two year old retorted when his father called him to account: "Why did you hit John?" "I hit him 'cause he hit me!"

Freud reminisces concerning his relationship with his father. They would go into the "beautiful forest" together; and as soon as the little boy could walk, he would attempt to toddle off on his own. He grew to admire his father as the wisest, wealthiest and most powerful man he knew. One striking incident impressed in Freud's mind is when impelled by sexual curiosity, he intruded into his parents' bedroom only to be driven out by his father's emphatic command.

Although Freud made little mention of his mother in the story of his childhood, there definitely was a mutual, deep attachment between mother and son.

When the boy was three years old, the family moved to Leipzig. A year later, the Freuds settled in Vienna. Here Freud was to spend almost the entirety of his life. For a long time, the family lived in Kaiser Josef Strasse, now renamed Heine Strasse. Freud stated that the name Josef always played a great part in his dreams, his view being that in dreams of emperors and kings these potentates symbolize the father. Joseph II (for whom the street was named) had been regarded by the liberal bourgeoise as the finest flower of the Hapsburg dynasty; as an exemplar of wisdom, benevolence, progress, and devotion to duty. In reality, this emperor was a despot who paid lip-service to the ideals of the French enlightenment. His progressiveness has been termed by Wittels as "ill-digested Voltairism", his benevolence was capricious, and his wisdom was but a fable. His notion of freedom was embodied in the maxim: Everything for the people, nothby the people. Long residence, during the impressionable years of boyhood in a street whose name carries such association was bound to have a strong influence on the highly imaginative youngster. Freud became an emperor, one around whom legends began to accumulate, who holds enlightened but absolute sway in his realm, and is animated by a rigid sense of duty—he became a despot who tolerated not the slightest deviation, held council behind closed doors, and was advised by no one—again he became the warrior entering battles against seemingly overwhelming odds, yet losing not a single encounter.

In his everyday life, little Sigmund searched for an exalted personage to set up on a pedestal. The natural figure for the boy was his father, and in this aspect two incidents in his life remained emblazoned in the boy's memory. The first occurred when Freud was eight years old. Young Sigmund had just disobeyed his father. The father turned and remarked to his wife, "The boy will never amount to anything." Little Sigmund on the verge of tears blurted out, "Your predictions will not come true, and I will be able to prove it!" The second incident occurred a few years later and here strong hints can be found of Freud's disappointment that he did not have a more powerful and successful father. When Sigmund was ten years old, his father began to let the boy accompany him on walks and laid open before him in conversation his opinion about world affairs. Intending to show how improved were the times in which the youngster was born the father told him a tale on one of these occasions: "Once when I was young, I went for a walk in your birthplace; I was well-dressed with a new fur cap on my head. A Gentile steps in my way, strikes with one hit my cap into the dirt and calls out: Jew, get off the sidewalk!" Sigmund leaned forward eagerly awaiting strong and drastic action on the part of his father. "What did you do, father?" "I stepped into the middle of the street and picked up my hat" was the quiet answer. Years later, in The Gesammtausgabe, Freud wrote, "That did not impress me as sufficiently heroic for the great and strong man who led me by the hand."

Freud attended the Sperl Gymnasium and throughout the eight years of his studies was the leader of his class; he enjoyed special privileges there and was required to take few examinations. Although the family lived in very limited circumstances, his father insisted that, in the choice of a profession, Freud should follow his own inclinations. For a long time he hesitated in his choice of a career between law and natural science. His skill as a speaker, his eloquence, his interest in universal history and in humanism, seemed to mark him out for the study of the abstract sciences. Shortly before leaving the Gymnasium he resolved to become a medical student but in his "Autobiographical Study", he states that he felt no particular predilection for the career of a physician. Rather was he impelled by curiosity, which was, however, directed more towards human concerns than towards natural objects. Under the powerful influence of a school friendship with a boy many years his senior, who grew up to be a well-known politician, he had developed a liking for law and social activities. At the same time, the theories of Darwin were beginning to take the stage, and to Freud, they held out hopes of an extraordinary advance in the understanding of the world; but he writes that it was not until he became acquainted with Goethe's "incomparably beautiful essay on Nature," that the enthusiastic desire to become a doctor was awakened.

On leaving the Gymnasium, Freud went to England to visit his half-brother who was twenty years older than himself. This journey greatly expanded his outlook. In Austria, he had never been able to escape the sense of inferiority which early affected him, as it does all Jews in German-speaking lands, and especially those who move in intellectual circles. In England, Freud renewed acquaintance with members of his family who had not only escaped this danger, but also had fared much better financially. Furthermore, conversations with his half-brother gave Sigmund a fuller and more affectionate understanding of their father.

In 1873, he matriculated at the University of Vienna. At that date, the medical faculty was at the height of its fame. Brücke was professor of physiology from 1849-1890, and was one of the teachers who, in conjunction with Hyrtl, Nothnagel, and Rokitansky, established the theoretical foundations on which the great Viennese physicians were to build their practical successes. Incidentally, it was Nothnagel who concisely stated, "Whoever needs more than five hours of sleep should not study medicine. The medical student must attend lectures from eight in the morning to six in the evening. Then he must go home and read until late at night." The scientific investigators of this epoch had an amazing competence for the observation and description of natural phenomena, and among them Brücke was preeminent.

Upon his entrance into the Medical School, he encountered many disappointing matters. He writes: "When in 1873 I first joined the University, I was met by some appreciable disappointments. Above all I found that I was expected to feel myself inferior and an alien because I was a Jew. I refused absolutely to do the first of these things. I have never been able to see why I should be ashamed of my descent . . . of my race. I put up without much regret my non-admission to the community for it seemed to me that in spite of the exclusion, an active fellow worker could not fail to find some nook or cranny in the framework of humanity." These first impressions at the University, however, had one consequence which was afterwards to prove important, for at an early age he was made familiar with the fate of being in the opposition and of being put under the ban of the "compact minority."

In 1876 Freud entered Brücke's laboratory where he worked happily for nearly six years. Here he found rest and satisfaction— and men, too, whom he could respect and take as his models; the great Brücke himself, and his assistants Sigmund Exner and Ernst von Fleischl-Marxow. In Fleischl-Marxow, whom Freud considered brilliant, he found a warm friend. In 1877, in Brücke's laboratory, he worked out a problem in neuro-anatomy, on moammocoetes, a larval form of an early vertebrate and reported on his studies of the nervous system of the fresh water crab. In 1939 Jelliffe wrote concerning this: "Thus Freud may correctly be said to have grasped the significance of the neurone theory network from his studies of the living tissues of the fresh water crab." During the years in which he worked in Brücke's laboratory, Freud was decidedly negligent in pursuing his medical studies, and it was not until 1881 that he somewhat belatedly received his degree as a Doctor of Medicine. The various branches of medicine proper had held no attraction for him; psychiatry had been his sole genuine interest.

Freud considered the turning point in his career to be 1882, when Brücke advised him to abandon his theoretical career. This advice apparently was given because of Freud's poor financial position. So the twenty-six year old Freud left the physiology laboratory to enter the General Hospital in Vienna as an "Aspirant." He worked in various departments, and, for more than six months, studied under Meynert whose work and personality had attracted him while he was still a student. As the subject of his work, he chose the medulla oblongata. He became an active worker in the Institute of Cerebral Anatomy. He wrote papers upon the course of the tracts and the nuclear origins in the medulla oblongata. But from a practical viewpoint, brain anatomy was no better than physiology, and, with an eye to material considerations, he began to study nervous diseases. In this branch of medicine, there were few specialists in Vienna. In the course of the following years while continuing to work as a junior physician, he published a number of clinical observations upon organic diseases of the nervous system. In his Autobiographical Study, Freud's discussion of this period of his life gives us a revealing incident worthy of quotation. Freud wrote: "I was able to localize the site of a lesion in the medulla oblongata so accurately that the pathological anatomist had no further information to add; I was the first person in Vienna to send a case for autopsy with a diagnosis of polyneuritis acuta. The fame of my diagnosis and of their postmortem confirmations brought me an influx of American physicians, to whom I lectured upon the patients in my department in a sort of pidgin-English." This passage is indeed valuable as a true insight into the man, for while Freud's disciples, biographers, and others have made so much of his modesty, we see that he was not without some vanity.

On one occasion, Freud introduced to his audience a neurotic suffering from a persistent headache as a case of chronic localized meningitis; his audience rose in revolt, and his premature activities as a teacher came to an end. Freud's unfortunate episode may be partially excused when we realize that the greatest authorities in Vienna at that time were in the habit of diagnosing neurasthenia as cerebral tumor.

It was about this time (1884) that an amusing, although not insignificant, happening occurred in young Freud's life. A side interest had led him to obtain from Merck some of what was then the little-known drug, cocaine, to study its physiologic activity. While this research was in progress an opportunity presented itself for making a visit to his fiancée from whom he had been parted two years. Quickly the investigation of cocaine came to an end, and in his book on the subject, Freud was content in prophesying that further use for it would soon be found. When Freud returned from his holiday, he found that one of his friends, Carl Koller, to whom he had mentioned the possibility of cocaine's being useful in the anesthization of the eye, had made the decisive experiments upon animals' eyes and had demonstrated them at the Ophthalmological Congress at Heidelberg. So Koller became the discoverer of local anesthesia by cocaine, which has now become so important; and Freud had moved a little closer to making the cause of his journey his wife.

In the spring of 1885, Freud was appointed lecturer on neuropathology and at the same time was awarded a traveling fellowship. At last he was to go to Paris and study under the brilliant Charcot! So in the autumn of 1885, he became a student at the Salpetriere in Paris. One day he overheard Charcot express his regret that since the war he had heard nothing from the German translator of his lectures. That night, Freud wrote Charcot offering to undertake the translation. Charcot accepted the offer, and Freud was admitted to the circle of his personal acquaintances, and from that time forward took a full part in all that went on in the Clinic.

What impressed young Freud most during his studies under Charcot was the latter's investigations upon hysteria. He had proven the genuineness of hysterical phenomena and their conformity to laws, the frequent occurrence of hysteria in man, the production of hysterical paralysis and contractures by hypnotic suggestion, and the fact that such artificial products showed, down to their smallest details, the same features as spontaneous traumatic attacks. Charcot proved beyond a doubt that ideas can induce bodily changes. These views came into sharp conflict with the then dominant views in Germany. It is quite probable that Freud was not slow to realize that he was learning something which would bring him into sharp collision with the Viennese school. Before leaving Paris, Freud discussed with the great man the hypothesis that the hysterical paralyses and anesthesias of the various parts of the body are demarcated according to the popular ideas of their limits and not according to anatomic facts. Charcot took no special interest in this point. Today we know that Freud had hit upon the cornerstone of localization in hysteria.

After about a year in the Salpetriere, Freud returned home by way of Berlin. In Vienna, he settled down as a specialist in nervous diseases. His attempt to propagate the teachings of Charcot met with a bad reception. Even his old sponsor, Meynert, ridiculed the ideas and suggested to Freud that he find some cases in Vienna similar to those he had described. Freud tried to do so; but the senior physicians, in whose departments he found the desired cases, refused to allow the cases to be observed or worked with. Freud recounted how one of them, an old surgeon, broke out with the exclamation: "But, my dear sir, how can you talk such nonsense? 'Hysteron' means the uterus. So how can a man be hysterical?" Finally, outside the hospital, he came upon a young man with a case of classical hysterical hemi-anesthesia, and demonstrated it before the Vienna Medical Society. This time, he was applauded, but with the ending of the applause, was also forgotten. The impression that the high authorities had rejected his innovations was quite evident. Soon afterwards, Freud was excluded from the laboratory of cerebral anatomy and for a whole session had nowhere to deliver his lectures. He withdrew from academic life and ceased to attend the learned societies. In later years, Freud was often strongly invited to attend the Vienna Medical Society meetings, but he never again visited the organization.

In the autumn of 1886, Freud married Martha Bernays, the girl who had been waiting for him in a distant city for more than four years. Now Freud attacked the problem of making a living. His therapeusis consisted of two weapons, electrotherapy and hypnosis. Soon, however, Freud put aside the electrical apparatus as being totally ineffectual.

With hypnotism the case was better. While still a student, Freud had attended a public exhibition given by Hansen the "magnetist", and had become firmly convinced of the genuineness of the phenomenon of hypnosis. In Paris he had seen hypnotism used freely as a method for producing symptoms in patients and then removing them again. Then the news had reached Vienna concerning the school which had arisen at Nancy which successfully utilized suggestion with or without hypnotism. It was thus quite natural that during the first years of his private practice, Freud relied primarily on hypnotic suggestion in the treatment of his patients. He found hypnotic work to be really intriguing and the reputation of being a "miracle worker" quite flattering. In his work, however, he found two cardinal complaints: He could not succeed in hypnotizing every patient; some patients could not be put into a sufficiently deep sleep to produce somnambulism with amnesia which he considered indispensable for cures.

To improve his hypnotic technic, in the summer of 1889, he spent many weeks in Nancy. Here could be seen not only old Liebeault working with poor women and children of the working classes, but also Bernheim's astounding experiments with hospital cases. Two points deeply impressed him during his Nancy stay. The first was that there were mighty psychic forces hidden from human consciousness. The second was that some people cannot be hypnotized.

From the very beginning, Freud had utilized hypnosis aside from hypnotic suggestion to question patients concerning the origin of their symptoms. The inspiration for initiating this method is attributed to

Dr. Josef Breuer who was one of the most respected family physicians in Vienna. This man, who was of striking intelligence, and who had made important contributions upon the physiology of breathing and upon the organ of equilibrium, befriended Freud. Even prior to Freud's Paris studies, Breuer had told him of a case of hysteria which he had treated between 1880 and 1882 in a unique manner which had led him to penetrate deeply into the causation and significance of hysterical symptoms. Freud had been extremely impressed with Breuer's case material and in Paris had informed Charcot concerning the case. The latter took no interest in the subject; so the matter was, for the time being, put aside. Now that Freud was back in Vienna, Freud turned once more to Breuer's observations and made him recount more about the case. The patient, Anna O., had been a young girl with superior education and gifts, who had fallen ill while she was nursing her father of whom she was very fond. When Breuer saw the patient, she presented a variegated picture of convergent strabismus, disturbances of hearing, paralyses, numerous contractures, inhibitions and states of mental confusion. A chance observation showed the old physician that relief could be obtained from these clouded states of consciousness if she was induced to express in words the affective phantasy by which she was at the moment dominated. Breuer searched for a manner in which fully to utilize this factor. Finally he hit upon a plan. The patient was put into deep hypnosis and made to tell each time what it was that was oppressing her mind. With the overcoming of the depressive confusion attacks, the inhibitions and physical disorders were next rectified. In her waking state the girl could no more describe than other patients how her symptoms had arisen, and she could discover no link between them and any experiences of her life. In hypnosis, she immediately revealed the missing connection. It became apparent that all her symptoms went back to moving events which she had experienced while nursing her father, that is, her symptoms had a meaning and were residues or reminiscences of those emotional situations. In most instances, it turned out that there had been some thought or impulse which she had had to suppress while she was at her father's bedside, and that, in place of it the symptom had afterwards appeared. As a rule, the symptom was not the precipitate of a single such "traumatic" scene, but the result of a summation of a number of similar situations. When the patient recalled a situation of this kind in a hallucinatory way under hypnosis and carried through to its conclusion with a free expression of emotion, the mental act which she had originally suppressed, the symptom was abolished and did not return. By this procedure, Breuer succeeded, after long and painful efforts, in relieving the patient of all her symptoms.

Now to Freud the question arose as to whether or not it was possible to generalize from this single case. So Freud began to repeat Breuer's investigations with his own patients and eventually, after observing for several years, concluded that his findings were invariably confirmed in every case of hysteria that was accessible to such treatment. Freud proposed to Breuer that they should issue a joint publication. The latter finally agreed but only after vehement objections. In 1893 they issued a preliminary paper, "On the Psychical Mechanism of Hysterical Phenomena", to be followed in 1895 by "Studies of Hysteria", in which Breuer supplied the material content and Freud a theory of the unconscious and — for the first time — the importance of sexuality. Coincidences were noted between anxiety hysteria and coitus interruptus and between neurasthenia and masturbation, and the argument was drawn that actual neuroses were direct toxic effects of sex while the psychoneuroses were mental impressions of it. The medical profession greeted this book with stark incredulity. Even Breuer, who at first helped, broke with Freud when the latter pointed out the transference love in the patient, "Anna."

It was about this time that the Freud family encountered financial difficulties. Freud had sacrificed his flourishing practice to devote himself entirely to his new science. His professional income, the only means of making a living, dwindled rapidly just at the time when his family increased (six children, three boys and three girls were born within ten years). The attitude of friends and acquaintances in general was that of pity for the poor woman whose husband, formerly a clever scientist, had turned out to be a rather disgusting freak. Frau Professor, as Freud's wife was called by everyone, never wavered in her admiration. To her, he was a great man before a word of his book was written as well as afterwards. Her devotion was fully shared by the children. Friends of the family frequently made fun of the solemn way they spoke of everything concerning their father. For instance, it was said that when one of the children had been absent for some time and was met by another, the first words from the newcomer were, "Father now drinks his tea from the green cup instead of from the blue one." The life of the family is said to have revolved around the father as his life revolved around his work.

Mrs. Freud was small, lithe, active, the typical housewife who was always busy putting something in order or cleaning and brushing — always accurate to the last detail of neatness and order, yet in one respect she did not conform to the type. Instead of being regarded by her servants as a curse, as these model housewives usually are, she was extremely popular with them. Her great kindliness and deep humanity never tolerated the idea that the life of a human being should be subordinated to the welfare of the furniture. She showed much generosity in finding the most suitable Christmas presents for all the retainers of the family, not only for everyone who was connected with the household but also for his or her relatives. Her sister, Minna, once made the remark that the line was drawn at the niece of the milkman.

But to return more directly to Freud's work, we find that he began realizing the limitations and disadvantages in using hypnotism as a therapeutic measure. Searching for a substitute, he remembered an experiment he had witnessed while working with Bernheim. He recalled the awakening of a person from the state of somnambulism with the apparent loss of memory of what had happened while he was in that state. Bernheim had maintained that the memory was present all the same; and if he insisted on the subject remembering, if he gave assurance that the subject knew it all and had only to say it, and if at the same time he laid his hand on the subject's forehead, then the forgotten memories returned, hesitatingly at first, but eventually in a flood and with amazing clarity. Freud determined that he would act in the same way. So hypnosis was abandoned and replaced by free association. The only part of hypnosis being retained was that of requiring the patient to lie upon a sofa, while the physician sat behind, seeing the patient but not being seen. Using hypnosis, Freud had called it "the cathartic method" because he considered its efficacy to be upon the mental and emotional purging which the patient went through during the treatment. The symptom was considered a monument of some disagreeable and forgotten (repressed) act of the patient's past. The patient did not, however, recognize the significance of this monument any more than a child could understand Einstein's theory of relativity.

This idea for the first time showed the importance of distinguishing between conscious and unconscious states and later was amplified and developed as the psychology of the unconscious. The patient was supposedly unable to give expression to his feelings because the situation in question precluded it, so that the idea was repressed from consciousness and thus excluded from further elaboration. But as the repressed material remained dynamic and continuously strove to come to consciousness, the sum of energy finally worked itself to the surface on a wrong path and thus produced the symptom. In other words, the symptom represented a conversion of psychic energy into a physical manifestation, such as pain or paralysis or even into an obsession. The cure or the mental and emotional catharsis was effected through the process of abreaction, that is, the hypnotized patient abreacted or worked off the repressed material by living through it and giving free vent in speech and action to the feelings that were originally excluded from consciousness.

Some have said that Freud was as much an artist as a scientist in the birth of psychoanalysis. True and therefore all the more power to him! For who but an artist would grasp by intuition many matters that would have escaped an investigator rigorously bound to traditional scientific methods. The founder of psychoanalysis, he would never have become the elaborator of it that he was, if he had proceeded cautiously.

Many consider Freud's work and psychoanalysis the typical product of Vienna and its peculiar moral atmosphere; the same has been said of Franz Schubert's music. Freud attempted to explode this allegation in the "History of the Psychoanalytical Movement" by pointing out the logical fault of the casual connection between his discovery and the asserted looseness of sexual morality in Vienna. From a science which had been hushed, trampled on and debauched, psychoanalysis has grown until now its broadening fields may be considered to be the foundation of modern psychiatric thought.

The basic concept of psychoanalysis centers about mental topography which considers the mental apparatus as an integrated whole of which three concrete parts may be described. The first part is the id, entirely unconscious harboring the crude instincts seeking expression. The instincts differ considerably from the conscious and are essentially selfish, uncivilized, and unmoral. The second group is termed the ego. The ego is split off from the id by contact with reality through the sensory-motor apparatus whose function is to maintain harmony between the id and external reality. It is the conscious self. The last group is the super ego, largely conscious, the repressing agency, whose function is to keep id under cover. It might well be termed the inner conscience or censor and embodies the ideals, morals, and inhibitions characteristic of man.

There are many other points to the psychoanalytic theory, but Freud considered three to be paramount: the libido, infantile sexuality, and transference.

The libido: he believed that most mental processes are derived from the interplay of forces which are originally instinctive in nature. These forces represent themselves by images or ideas which carry an emotional charge. They are termed the libido or sexual instinct and largely determine the quality of the personality structure and functioning, the principal types being dominantly in chronological order of appearance narcissistic, oral, anal, phallic, and genital.

Infantile sexuality: sexual development of the child goes through many phases before normal adult development is reached. Freud applied the term, "Oedipus complex," to immature sexual attachments which persist into adulthood. He believed that the infant develops emotional attachments with strong sexual coloring toward the parent of the opposite sex. Normally at puberty, other interests replace the parental attachment. With the failure of this displacement to occur the individual may be immature and unable to make a satisfactory adult sexual adjustment. Partial frigidity in women or relative impotence in men often occurs.

Transference concerns the emotional relationship between the patient and his physician. Especially in psychoanalytic therapy is this important. It may be affectionate or hostile, or passive or active. It is believed to be based not upon the actual situation, but instead comes from the emotional relationships of the past, that is, the patient tends to identify the doctor with his parents and other important characters of the past, transferring to the physician the feelings which he formerly had for the original character.

Modern psychoanalytic therapy consists first of a series of interviews usually arranged to determine whether or not the patient is suitable and cooperative. If the patient accepts treatment, the analytic procedure may be commenced. This often requires one hour sessions (four to six times weekly) over a period ranging from one to five years. During a session, the method of free association is used, the physician largely guiding the patient.

With a successful analysis, the patient should have recovered from the mental conflicts and should understand how they develop and be able to prevent their recurrence. It should be pointed out, however, that some, particularly the poorly organized, are apparently made worse.

To think that Freudian psychoanalysis is the only type is indeed a most serious error. Jungian psychoanalysis as promulgated by C. G. Jung of the University of Zurich has come to play a very important role in psychiatric thought. Jung was an early follower of Freud, but in 1913 they parted "without feeling the need to meet again" and since that date, there has been an active warfare between the two schools as well as an increasing divergence in theory and practice. The Jungians' main argument is against the Freudian pansexuality of human interests. Jung deals with a personal unconscious which harbors the repressed desire of the individual and also with a collective unconscious in which is preserved the experience of the race. The collective unconscious is inhabited by divinities, saviors, magical beings and other strange powers termed arche-types whose activities dominate the fanciful world as expressed in mythology, folklore, dreams and psychoses. Still another school is that of Alfred Adler, another of Freud's ex-followers. Adler developed what he called "Individual Psychology" as an outgrowth of Freud's ideas. This places less emphasis on Freud's libido theory, and emphasizes more the individual's striving for power, superiority, and self-assertion. It stresses the theory of organ inferiority.

Between and around these three schools, one which might be termed realist and the other two romanticist have developed and will continue to develop many tangential lines of thought.

For several years Freud had been studying the use of dreams in psychoanalysis. Early in 1900 appeared "The Interpretation of Dreams." Now the basic structure of psychoanalysis was complete. The "Interpretation of Dreams" was fiercely attacked. Many close followers shook their heads over this book.

Let us examine Freud's beliefs: he believed the dream related by the dreamer to be not the dream that was dreamed. To learn the hidden significance of the dream, it was necessary to reach back from its ostensible meaning to the thoughts of which it is the mask. Like a cipher, the dream can only be understood with the aid of a key. The deciphered dream was invariably associated with a deep significance. Furthermore, Freud believed that the dream gave expression to impulses that were deeply hidden. Forbidden wishes, feelings of love and hate, criminal trends, delusions of grandeur, self-adulation, longing for death — all these manifest themselves in dreams, but their meaning is so artfully covered up that the dreamer is unable to grasp the significance of his own dream. With an understanding of dreams, we find unwelcome thoughts. The dead return—the suppressed thoughts of evil, the shapes that we want to keep from the light of the day, the thoughts that had been thrust down into the well of the unconscious. In our dreams, we murder our nearest and dearest. We revel in perversions at which we shudder in our waking hours. All our criminal instincts are unchained in the dream. No one knows what we dream. We do not even know it ourselves, for the dream distortion hides from us, as from others, the true meaning of our orgies. Dreams that might betray us too readily are forgotten; they cannot pass the censor. Now, how did

Freud utilize these ideas? Very simple—the munition manufacturer dreams of war; the henpecked husband dreams of free love; the woman whose husband is impotent dreams of athletes and operatic tenors. The wife who murders her husband in dreams makes up for it by lavishing so much attention on him that the poor fellow is driven almost crazy. The henpecked husband dreams of obscene adventures, and atones for his dreams in daily life by sanctimoniousness, by looking down upon the most innocent pleasures. Little wonder that the world frowned and has continued to frown at this work on dreams. In Vienna this book was met with complete silence and relegated to the care of a clinic assistant who had already written a book from the opposed viewpoint.

Nevertheless, a little circle of physicians had begun in 1902 to gather around Freud into which came Dr. Alfred Adler, in 1906 Dr. C. J. Jung, and in 1907 Dr. Eitinger. Then in 1908 a first psychoanalytical congress was held at Salzburg, with Jung being made editor of the Journal. The consequences concerning Jung's ascension to power are indeed most interesting.

Freud preferred to leave all formal functions to others; filling an official post, being the figurehead, standing personally in a prominent position were things that went strongly against his grain. For this reason in 1910 he insisted on the installation of Jung as president of the newly founded International Association, which later proved to be a costly mistake. The Zentralblatt was launched with Adler and Stekel as editors; and, later to deal with the applications of analysis to the mental sciences, Imago, edited by Hanns Sachs and Otto Rank.

The years 1904-1908 found Freud working primarily on sexual theories. The lecture on Modern Sexual Morality and Modern Nervousness was heard around the world. Quickly, he was labeled a pansexualist. Psychiatrists and laymen alike jeered at the Austrian extremist. For psychoanalysists who went further into the question, a sexual dilemna was raised. For, on the one hand, psychoanalysis had shown that sexual abstinence was one of the most important factors in the formation of neuroses. On the other hand, the economic circumstances of most students made it impossible for them to marry early. Morality forbade the seduction of young girls, the danger of infection made sexual intercourse with prostitutes inadvisable. Finances and judgment made early marriages not to be desired, and so Freud's advice to the young students was, "Be abstinent, but under protest." He believed that it was imperative to keep alive the inner protest against a social order which prevented mature young men from fulfilling a normal instinctive need. Like Anatole France whose writings he loved, Freud did not believe in sudden and violent revolutions; he put more faith in the steadily mounting, continuous force of patient resistance to bring about ultimately changes in the social order. He often reiterated that in regard to money and to sex, men are hypocrites; in both these realms they refuse to confess their true needs. Very illustrative of this theme and yet very amusing is a passage from a novel of Anatole France. In Monsieur Bergeret a Paris, a young man attempts to seduce a lady. Anatole France, the connoisseur of women concludes his description as follows: "He came to her again, took her in his arms, and covered her with caresses. Within a short time her clothes were so disarranged that — aside from any other considerations — shame alone compelled her to disrobe."

Sensation-hunters had hoped to find in his life all the wildness that they did not like to see in their own mind, and were sorely disappointed to find out that Freud's way of living showed no pan-sexualism. His thoughts and desires were too well-balanced.

Nineteen hundred nine saw the unsuccessful journey to the United States. An invitation from Clark University to give a series of lectures and to receive an honorary degree on the occasion of its twentieth anniversary was the first public recognition of his new science, and the lectures were to become an undisputed triumph. Besides, Freud was grateful for the courtesy shown him by Professor G. Stanley Hall, the President of the University, and he felt unmixed respect for Professor Putnam in Boston who, in spite of his Puritan background and ancestry had defended the "immoral" psychoanalysis. Accompanying Freud were three of his best friends at the time: Ferenczi, Jones and Jung. To Freud, it was like the realization of a fantastic daydream when he took the lecturer's seat in Worcester to give his five lectures on psychoanalysis. Psychoanalysis had ceased to be an illusion, it had become a valuable piece of reality. Yet everything went the wrong way and he came home with a heavy load of dreary impressions which henceforth distorted his picture of America. Years later he confided to a friend: "America is the most grandiose experiment the world has seen, but I am afraid it is not going to be a success." Although Freud had been successful in explaining analysis, he had been unhappy and physically sick. Freud often made disparaging remarks about America and Americans. He resented the overeagerness of the Americans to entertain him. Having a will of his own, he became disgruntled when he was rushed around from place to place to see things in which he was only moderately interested.

"American women," he said, "are an anticultural phenomenon. They have nothing but conceit to make up for their sense of uselessness. You have a real rule of women in America. You, your young men go to college with girls, fall in love and marry young at an age when the girls are usually much more mature than the men. They lead the men around by the nose. Your average American man approaches marriage without any experience. You wouldn't expect a person to step up to an orchestra and play first fiddle without some training, but the American man steps into marriage without the least experience for so complicated a business. But," he continued, "your American woman has her good points, too; she hasn't got the European woman's constant fear of seduction, for example."

September 1911 found the International Psychoanalytical Congress holding its third meeting. Jung was again reelected president — this time with Freud's opposition. The schism had started.

Nineteen hundred twelve-thirteen brought the two grave secessions. Adler started the previously mentioned school of "individual psychology" which repudiated the importance of sex, traced character and neuroses to body-zones and to organ inferiority, and emphasized the "masculine protest." Jung, Freud's "dear and trusted friend", started an abstract, impersonal and non-historical school of "analytical psychology", rejecting what he found repellant in Freud, especially infantile sexuality and the Oedipus complex.

Then the first world war came on the scene. Immediately Freud's concepts of the "advantage of being ill" and "flight into illness" came into great popularity. During the war, Freud's writings continued to be prolific. With unmixed pleasure he observed the extreme usefulness of his principles in the therapy of soldiers. With peace, he settled back to complete all the writings that lay in his mind, but in 1923, Freud's excursions and discussions came to a sudden end with the ominous appearance of disease — a carcinomatous growth inside the mouth. Until the outbreak of the fatal disease his vigorous vitality had been undimmed by age although he approached his seventieth year. The hope for a ripe old age, attained in good health and strength of body and mind was not unjustified. His mother had borne him, her eldest son, before she was twenty; she was still alive to see his seventieth birthday. She reached the remarkable age of ninety-three years. Until about a year before her death she was hale and hearty, and full of energy. The tale is told that on the evening before his seventieth birthday Freud went to see her and receive her congratulations, so that she should not have to come the long way from the suburb where she lived to his house.

Next morning the first visitor who rang the bell was his mother.

The first superficial operation (1903) performed by a former fellow student of Freud, Professor Hajek, had done nothing to stop the malignant growth. The best experts were called in, and a radical operation was decided upon. A portion of the jawbone had to be removed; eating and talking were made possible by prosthesis. Professor Pichler, the famed surgeon of Vienna took over the treatment and prolonged Freud's life for a number of years. Since the surface of the scar left by the operation in the mucous membrane of the mouth was constantly changing, the prosthesis had to be changed and remodeled constantly. In spite of all this it caused constant pain which Freud bore without complaint. But that he was extremely sensitive about the resulting speech defect which made it at times difficult to understand him, there can be no doubt. More and more, it became his habit to live in seclusion and to see, besides his family and his analysands, only his intimates or those in whom he was particularly interested. He was still more sensitive about being observed while eating, and invitations to dine with him became an extreme rarity.

The seventy-fifth birthday brought not only congratulations but also celebrations and acclamation. But the more the name of Freud was honored and the greater grew the number of those who wanted to express their reverence and admiration, the wider became the gulf between these loud glorifications and the suffering, withdrawn old man who, in his seclusion, continued his work with great determination while he saw death creep relentlessly nearer. It was known that Freud was fond of orchids, and orchids of all colors and descriptions came by the cartload. The modest conservatory, where his wife by extreme diligence had grown a few choice flowers, was crowded with orchids which overflowed into the living and dining rooms. Freud, however, paid little attention to them, and two or three days afterwards they were to be found in the garbage can — a fitting symbol of fame that comes late, extravagant and indiscriminant.

The world had become "Freud-conscious." The best and foremost of contemporaries sought to make his personal acquaintance: Einstein, Thomas Mann, Romain Rolland being among them. In Lou Andrea-Salome, the friend of Friedrich Nietzche and later in Marie Bonaparte, Princess of Greece, he found disciples who combined feminine intuition with the gift of clear and independent thought.

The place of the dwindling human contacts was taken by a new and unexpected companionship. His daughter, Anna, who loved dogs became the owner of a big Alsatian. He was a very nice and friendly dog, but somewhat big and noisy for an apartment. Freud, instead of protesting, became attached to the dog and enjoyed his pranks, spoiling him not a little. Not much later Marie, Princess of Greece, presented him with a Chow. This dog was the daughter of Tartoun who belonged to the Princess and united in his manner the dignity of a nobleman with the polished manners of a courtier. Freud became very fond of the dog and still more of its puppies. From then on, Freud was seldom seen without one of his dogs. In the midst of his talk and in spite of his pains he gave them some attention—watching them tenderly, superintending their food and drink, playing with them, and patting their heads; he used to play with his ring, which previously he had frequently removed from his finger and twirled on his desk.

So the years went by, each adding slightly to the triple load of old age, pain, and the threat of imminent death. Soon the middle age wiry man was changed into the picture of a bent, icy-grey, shriveled old man. But what counted most in his life did not undergo any change; his work, his gift for new and original thought, his insistence on distinguishing between truth and hypocrisy were still as much a part of him as the breath of life.

At the advanced age of eighty, Freud returned to the study of the Hebrew Bible (Old Testament) which had attracted him as a boy of ten, and he presented the world with his Moses and Monotheism, a book which many consider the most interesting Freud ever wrote. It was in his "Autobiographical Study" that Freud stated, "My early familiarity with the Bible (at a time almost before I had learned the art of reading) has, as I recognized later, an enduring effect upon the direction of my interest." These simple words have a deep meaning. Freud realized that the biblical myths, as myths in general, are expression of human urges and impulses, cravings and longings, that moved man in the infancy and childhood of the human race, that move every child in its impressionable years when he recapitulates man's mental development from the animal to the civilized stage. Man was supposed to be continually wrestling with these urges and impulses, often being overcome by them to the end of his life. In his mature manhood, Freud was to find a cure for many of the mental sicknesses caused by the conflict of those psychic forces and devise a method for the interpretation of the myths.

Nineteen thirty-three saw the rise of Hitler to power. When Freud learned that in Berlin his books, together with Heine, Schnitzler, Wasserman and many others, had been solemnly consigned to perdition and burned, he said calmly, "At least I burn in the best of company."

When queried as to his own fate at this time, he said, "They told me that psychoanalysis is alien to their Weltanschauung, and I suppose it is." He said this with no emotion and little interest, as though he was talking about the affairs of some complete stranger. A dark cloud hovered over Europe — soon Austria was marked as its next victim. Freud's situation became especially dangerous in the event of a German invasion. His books had been solemnly burned in Berlin, and his name was among the first on the proscription list. Offers of an asylum, a place of safety in a better-protected country had been made, but he refused to accept them.

Then the long foreseen happened: Hitler invaded Austria and the rest of the world stood by without lifting a finger. The brutalities committed during five years in Germany became but a trifle when compared to what was dealt out in the first few days of the occupation. With the news of the invasion, Ernest Jones came from London and Marie, Princess of Greece, from Paris. These together with Mrs. Dorothy Burlingham of New York, who had lived in Vienna almost as a member of the Freud family, formed a sort of bodyguard by using their influence with the diplomatic representatives of their respective countries. In this way Freud was spared the personal insults and injuries which were inflicted on thousands of his race and many others.

Soon Freud was invited by the English government to take his residence in England. The permit was finally granted but only after the Nazis had confiscated everything they could lay their hands on, and Anna Freud had been given the third degree at Gestapo headquarters in an attempt to get a confession from her as to how and when money had been sent out of the country or otherwise concealed. By the sacrifice of a large sum of money, Princess Marie had succeeded in buying back Freud's collections and his library which accompanied him to England. Everything else Freud had was confiscated.

On his way through Paris, Freud was greeted by the American ambassador to France, Mr. Bullitt; in England he was warmly welcomed. A great deal of publicity was given to his arrival. As soon as possible he escaped from it and returned to his work in the old persistent, unostentatious way as if nothing extraordinary had happened to bring him from Bergasse 19 to 20 Moresfield Gardens. Here the atmosphere appeared quite different from the surrendered country he had just left. The house was pervaded by a peaceful and calm atmosphere. The rooms were much more comfortable and airy than the little study in Vienna. The garden was not large, but it had a fine lawn, and old trees jutted over the walls. But behind this superficial curtain of tranquility stalked the dreaded reality, the presence of constant pain and suffering. The pain in his mouth permitted him to find but little sleep. Occasionally, he slumbered for a few minutes in a swing couch that had been constructed in the garden. Here he could be seen resting, sometimes in light slumber, sometimes caressing his Chow who did not leave his side for a moment. He appeared unbelievably old and very ill. Every word was pronounced at the cost of an enormous effort. But these tortures did not wear down his will. His analytical hours were kept whenever he had even the slightest alleviation of pain, and when he was strong enough to hold the pen, his letters were written with his own hand.

Not once did he complain. No moan or lamentation came from his lips. But he did refuse then, as he always had, to lighten his burden by self-deception or self-willed ignorance. Unflinching without even using the emotional refuge of self-dramatization, which is so often utilized, he met his fate.

On September 23, 1939, a great man passed down the aisle of time — Freud the discoverer, to his last days the courageous adventurer in the realm of causations of human behavior.

Above even the clamor of war was heard the news of the passing away of the great psychologist. The position he occupied in science and mental therapy will scarcely be filled by any one successor. Psychology's vocabulary is full of words which testify to his influence, and a compilation of his works shows twelve complete volumes and 179 titles. Yet what to mankind was a tragic loss was to the man himself a release from a life filled with sufferings that were intensified in the last years and became unbearable at the very end.

Much discussion has occurred about the place that Freud and his discoveries will take in the history of human thought. No one can doubt the eminence of his originality. Over the years he had vitalized whole sections of thought in many fields—psychiatry, psychology, sociology, education, social work. Even the philosophers took note of his clinical observations and theories. Indeed, it would be quite difficult to hazard a guess as to which field Freud's contributions will ultimately prove to have the greatest value. At any rate, in whatever category Freud and his genius can and will be placed, the world's thought is enormously indebted to him.

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A NATIONAL HEALTH DEPARTMENT

The Council on Medical Service of the American Medical Association is to be congratulated on the news bulletins with which they are keeping the members of the organization informed as to legislation in Washington.

Bulletin No. 6 discusses the establishment of a separate national health department. The readers of the Journal of the A. M. A. who have been following Fishbein's history of the organization which has been coming out serially in the Journal will remember that almost sixty-five years ago the A. M. A. called for the organization of a separate department of health under a cabinet officer. A few years later a committee was appointed to bring this matter before Congress and almost continuously up to the present time, the House of Delegates has recommended the establishment of "A Federal Executive Department to be designated as the Department of Health, with a doctor of medicine at its head who shall have general supervision and direction of the affairs of the federal government pertaining to the health of the people."

It appears possible that this much to be desired department might be established. There are two bills before the Senate at the present time; one, No. 140 and one, No. 545. The latter provides for a separate department of health with a cabinet head, the other, the establishment of a rather heterogeneous group, consisting of three executive departments; welfare, education and medical activities. The head of this division should have cabinet status and under him would be three executives for the three divisions which are represented.

It will be interesting to see what action will be taken by the House of Delegates at the coming meeting of the American Medical Association in June. There seems to be a considerable opposition of the medical profession to S. 140, as well as to S. 712. The bulletin of the Council on Medical Service states that if it is not possible or feasible at the present time to have a separate department of health it would be better to group health activities in a separate bureau and not have them tied in with other activities in a department with cabinet status.

THE DIGITALIS GLYCOSIDES

The tendency seems to be at the present time for a greater and greater use of the purified crystalline digitalis derivatives as a substitute for the powder or the tincture of foxglove. The powdered whole leaf has, up until a comparatively few years ago, proved reasonably satisfactory for the treatment of the patient with heart disease who must take this drug. However, there are certain advantages in the crystalline digitalis derivatives which make for greater exactness in its dosage and a more satisfactory maintenance of the digitalization. Here in the south, LaDue of the Department of Medicine of Louisiana State University was one of the first to urge the use of Cedilanid, which is a crystalline form of glycoside C which comes from *Digitalis lanata.*

This particular preparation is of particular value in the treatment of cardiac emergencies. On the other hand it is not absorbed when given orally with the same regularity as is Digoxin or Digitoxin. Consequently the last two preparations are probably to be preferred in the treatment of the ordinary ambulatory patient who requires digitalis. One of the advantages of Digoxin or Digitoxin is that the dose is given in the metric system and by weight. With these glycosides of digitalis the unsatisfactory method of standardizing a batch of digitalis leaves is no longer necessary. The dosage is exact and at a definitely predictable relationship to the human being. A milligram truly represents the same amount of effective material rather than a milligram of powdered leaf which contains a variable amount of the effective substance. The maintenance dose of Digoxin, incidentally after digitalization, is approximately 0.25-0.75 mg. daily. The dose of Purodigin, Cardiodigin, Crystodigin, and other brands of Digitoxin, 0.1-0.3 mg. to be taken once a day.

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The recent meeting of the New Orleans Graduate Medical Assembly was well attended by a group of physicians who came from all over the United States. There was a total registration of 1598 and a state representation of 33 different states. Louisiana, of course, led all of the states but there were 107 doctors from Texas, 51 from Mississippi, 36 from Alabama, 33 from Georgia, and the other sates of course having a smaller number in attendance. Incidentally California had three people attending and the little state of Vermont one. The two foreign representatives came from, in one instance, Canada and the other the Philippines.

The sessions of the meeting were very well attended, largely due to the fact that an excellent scientific program had been provided for the doctors who attended the meeting. This yearly meeting is growing, and growing rapidly in importance and in popularity. Undoubtedly there would have been many more in attendance if it were not for the hotel situation in New Orleans.

Following the meeting some of the attendants made a trip to Mexico City where a three day program of scientific import was provided for the visiting physicians. The scientific meetings were not too long so that there was ample time for sightseeing and for having a good time.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

EXECUTIVE COMMITTEE

March 29, 1947

At a meeting of the Executive Committee held in New Orleans on the above date considerable discussion took place concerning the present status of the EMIC program. Unfortunately, some of the doctors of the state have continued to accept cases under this plan although it was officially discontinued by the State Society in May of last year. Explanation has been secured from time to time from the Department of Health to the effect that when a doctor states he will accept a case the Department of Health will continue to participate in the plan by accepting the application also. In other words it appears that the only way to terminate the program is for ALL of the doctors to cooperate with the State Society by refusing to handle such cases under the EMERGENCY PLAN. As a result of this discussion the secretary was requested to secure a complete report from the State Department of Health as to the present status of the EMIC plan, including names of doctors who have continued to participate, and report the information secured to the House of Delegates.

The Congressional Committee has gone on record as opposing Senate Bills 140 and 712 and has so advised congressmen in Washington and the Executive Committee, at this meeting, approved action of the committee in this regard.

In discussion of the report of the Committee on Voluntary Prepayment Medical Insurance the question was raised as to the necessity for laymen on the Board of Directors of the Louisiana Physicians Service, Inc., and it was agreed by the Executive Committee that recommendation for such additions to the Board be made to the House of Delegates.

Resolution concerning Sections on the General Practice of Medicine, as approved by the American Medical Association was presented to the Committee and it was decided that this should be referred to the House of Delegates for discussion and action.

There has been opposition expressed during the past few months by members of the Executive Committee to the appointment of A. W. Dent to the Federal Hospital Council and although letters have been sent to the Social Security Administration concerning this subject there has been no action taken to rescind the appointment. The Executive Committee has therefore, in accordance with action at the meeting on the above mentioned date, brought the attention of this appointment to other state medical societies in the southeastern section of the country and the Southeastern Hospital Association.

The recommendations of the Committee on Budget and Finance, including approval of the budget as prepared for 1947, transfer of balance in the Cancer Committee checking account to the checking account of the general fund, investment of $10,000.00 of the amount in the checking account of the general fund in U.S.A. Series G Bonds and recommendation to the Chairman of the Committee for Aid to Indigent Members that $2,000.00 from the Fund for Aid to Indigent Members be invested in the same type bonds, were approved. Since the amount in the trust fund for Medical Defense will soon reach, by per capita appropriation, $20,000.00 it was decided that recommendation should be made to the House of Delegates that this fund be allowed to increase to $40,000.00. The subject of deficits which might occur by committees on arrangements for annual meetings was discussed and it was decided that any such deficits should be approved by the House of Delegates or the Executive Committee before being paid from the general fund. The Budget and Finance Committee was advised that it was the wish of the Executive Committee that a report of receipts and expenditures for 1946 and a copy of the 1947 budget should be sent to each member of the Society.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

May 12. Joint Scientific Meeting with the Louisiana State Medical Society, Roosevelt Hotel, 8 p. m.
May 13. Louisiana State Medical Society Meeting, Roosevelt Hotel.
May 14. Louisiana State Medical Society Meeting, Roosevelt Hotel.
May 15. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Veterans Administration Hospital Staff, 8 p. m.
May 16. The Orleans Society of X-ray Technicians, 7:30 p. m.
Lakeshore Hospital Staff, 8 p. m.
Executive Committee, Hotel Dieu, 8 p. m.
May 19. Hotel Dieu Staff, 8 p. m.
May 20. I. C. R. R. Hospital Staff, 12:30 p. m.
Charity Hospital Medical Staff, 8 p. m.
May 21. Charity Hospital Surgical Staff, 8 p. m.
May 22. DePaul Sanitarium Staff, 8 p. m.
May 23. L. S. U. Faculty Club, 8 p. m.
May 27. Baptist Hospital Staff, 8 p. m.
May 28. French Hospital Staff, 8 p. m.
May 29. Clinico-pathologic Conference, Touro Infirmary, 12 noon.
May 30. Poliomyelitis Preparedness Conference, St. Charles Hotel, 2 to 5 p. m. and 7:30 p. m.
New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.
May 31. Poliomyelitis Preparedness Conference, St. Charles Hotel, 9:30 p. m.
June 2. Board of Directors, Orleans Parish Medical Society, 8 p. m.
June 3. Eye, Ear, Nose and Throat Staff, 8 p. m.
June 4. Mercy Hospital Staff, 8 p. m.
June 5. Executive Committee, Baptist Hospital, 8 p. m.
June 6. Ochsner Clinic Staff, 8 p. m.

Every Thursday, Medical Toastmasters, Hutchinson Memorial Auditorium, 8 p. m.

N. J. Tessitore, M. D., Secretary.

NEWS ITEMS

Dr. Francis T. Gidman was recently certified by the American Board of Surgery.

Dr. Woodard D. Beacham and Dr. Thomas Benton Sellers attended the Mid-Year Clinic of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons in Philadelphia, March 7-8.

Dr. Marshall Michel delivered a paper on Acute Malignant Obstruction of the Colon at the Southeastern Surgical Congress meeting in Louisville, March 10.

Dr. J. Kelly Stone was recently installed as president of the Southern Baptist Hospital staff; he succeeds Dr. John T. Sanders. Other officials installed were: Dr. W. Robyn Hardy, vice-president; Dr. E. Garland Walls, secretary; and Dr. Joseph Wells, treasurer.

The Medical Toastmasters no longer meet at the New Orleans Athletic Club at 6:30 p. m. on Thursdays. They now meet in the Hutchinson Memorial Auditorium at 8 p. m. on Thursdays.

In the near future Mr. Rudolph O. Vorbusch will give a course in Parliamentary Law, which he has presented most successfully at the Young Men's Business Club and other organizations. The general membership will be invited to participate in this course, or to attend as guest.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THE RAPIDES PARISH MEDICAL SOCIETY

The regular monthly meeting of this organization was held on April 7 at the Hotel Bentley. The scientific program consisted of a very excellent talk by Dr. I. Epstein on "Early Manifestation and Diagnosis of Tuberculosis as Applied to the General Practitioner."

CHARITY HOSPITAL

A meeting of the Medical Division of Charity Hospital was held on Tuesday, April 15 at 8 p. m. in the Auditorium of the hospital. The program consisted of the following: A case presentation—whooping cough associated with possible heart failure by Dr. Ross C. Tilbury; a case presentation of a family epidemic of lead poisoning by Dr. David Spizer.

TOURO INFIRMARY

The regular monthly meeting of the Medical Staff of Touro Infirmary was held on Wednesday, April 9 at 8 p. m. The Department of Pathology presented a clinico-pathologic conference with a discussion by Drs. Roy Turner and Gordon McHardy. Dr. Albert Braunstein spoke about Pathologic Ophthalmic Changes in Diabetes Mellitus, and Dr. Irving Glassberg presented a case report on Polycystic Kidney and Ureteral Obstruction.

LOUISIANA STATE ALLERGY SOCIETY

On April 1 of this year a group of Louisiana allergists got together in Shreveport and formed the Louisiana State Allergy Society.

At this meeting Dr. Bernard G. Efron of New Orleans was elected president, Dr. Henry D. Ogden of New Orleans was elected vice-president and Dr. J. Dudley Youman, Jr., of Shreveport was elected secretary-treasurer.

A constitution was adopted at this same meeting.

The purpose of this society is to further the study of allergy, to promote discussion of the various aspects of allergy and to bring together the allergists of the state of Louisiana.

The meetings are to be held on the first day of the meeting of the Louisiana State Medical Society and in the same city as is the meeting of the Louisiana State Medical Society.

LOUISIANA TUBERCULOSIS ASSOCIATION

The Louisiana Tuberculosis Association held an institute for workers at the Jung Hotel, April 17-19. Among those appearing on the program were Drs. Julius Lane Wilson, John M. Whitney, Robert Smith, Sydney Jacobs and Maurice Campagna. Dr. O. P. Daly presided at the luncheon meeting Friday, April 18.

FACULTY APPOINTMENTS AT L. S. U.

The School of Medicine, Louisiana State University, announces the following faculty appointments:

Dr. Jerome T. Syverton, formerly associate professor of bacteriology, University of Rochester, as professor and head of the Department of Microbiology. In the past, the departments of pathology and bacteriology have been combined. Dr. Syverton will be responsible for the organization of a new department which will cover instruction and research in the basic factors in infectious disease, including bacteriology, virology, parasitology and immunology.

Dr. Curtis J. Lund, formerly associate professor of obstetrics and gynecology, University of Minnesota, as professor of obstetrics and gynecology on a full-time basis.

Dr. Harold L. Brauner, formerly instructor in pharmacology, Harvard University, as assistant professor of biochemistry, assigned to the Department of Pharmacology.

Dr. Leon Churney, formerly instructor in physiology, University of Pennsylvania, and recently released from military service, as assistant professor of physiology.

Dr. Allan L. Segal, formerly instructor in radiology, Columbia University, as clinical assistant professor of radiology.

Dr. James A. Wilson, formerly resident in obstetrics and gynecology, Charity Hospital of Louisiana, as instructor in obstetrics and gynecology on a full-time basis.

Dr. Harry D. L. Kaye, formerly resident in surgery at the New Haven Hospital and assistant in surgery, Yale University, as instructor in surgery on a full-time basis.

Dr. Philip B. Johnson, recently released from military service, as instructor in medicine on a full-time basis.

Other recent appointments include Dr. Juan E. Morales as clinical assistant in neuropsychiatry, Dr. Marie L. Surgi as assistant in pediatrics, and Dr. Robert M. Rose as clinical assistant in orthopedics.

NEW DIRECTOR AT CARVILLE

The USPHS announces that Dr. Frederick A. Johansen has been named Director of the National Leprosorium at Carville. Dr. Johansen has been connected with the Institution for some 23 years and undoubtedly is one of the most outstanding leprologists in the world. He has been a source of inspiration to the staff of the hospital and to the patients there.

Dr. G. A. Faget, the former Director of the Institution, has been sick for some months and it is our understanding that the Public Health Service will give him an assignment which should be less demanding on his physical well-being than the post he has now relinquished.

REFRESHER COURSE IN OBSTETRICS

At the request of the Louisiana State Department of Health, Section of Maternal and Child Health, the Tulane University School of Medicine will offer a Refresher Course in Obstetrics June 2-14, 1947. Registration will be limited so that each physician may receive individual attention.

Tuition for the course will be paid by the Section of Maternal and Child Health of the Louisiana State Department of Health. In addition, each physician will be allowed a per diem of $6.00 for the thirteen days' attendance at the course, for which a check will be mailed to him shortly thereafter.

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AMERICAN MEDICAL ASSOCIATION RADIO PROGRAM

The American Medical Association radio program entitled "Stephen Graham, Family Doctor" is being carried regularly on Monday night from 9:30 to 10:00 p. m. CST by all of the Mutual Broadcasting Company stations in Louisiana. These include KPDR, KCIL, KANE and WNOE. This program is very well worthwhile and can be heartily recommended not only to doctors and their families but to their patients as well.

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LOUISIANA CONFERENCE OF SOCIAL WELFARE

Under the presidency of Dr. C. L. Mengis, Director of the St. Landry Parish Health Unit, Opelousas, a three-day conference on social welfare was held at the Jung Hotel in New Orleans April 15-17. On the program appeared, among others, Mr. Charles Mitchell, Director, Health and Hospital Division, Office of the Governor, who discussed the state program. Dr. John A. Lewis, U. S. P. H. S., presented a paper on "The National Hospital Survey and Construction Program." Mr. Homer L. Hitt, of Louisiana State University, presented a paper entitled "Health in Louisiana." At a sectional meeting on mental health, Dr. Wilton E. Kirkpatrick discussed the National Mental Health Act. The section on physical health was presided over by Dr. Waldo Treuting and the topic "National Health Insurance Comes of Age—The Taft Bill", was presented. At the luncheon meeting of the Louisiana Legislative Council, the speaker was Dr. George W. McCoy, head of the Department of Public Health, Louisiana State University of Medicine, who talked on the subject "Some Newer Considerations in the Management of Leprosy." The following day a luncheon meeting of the Louisiana Tuberculosis Association was held at which time Dr. Julius L. Wilson presided and Dr. Sydney Jacobs, of New Orleans, was the speaker.

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DEDICATION OF THE FOUNDATION HOSPITAL

On Sunday, April 13 the dedication exercises of the Foundation Hospital were held at the former Camp Plauche Hospital. The Invocation was pronounced by the Archbishop of New Orleans, Most Reverend Joseph F. Rummel. Dr. Ochsner acted as master of ceremonies and addresses were given by Mr. J. Blanc Monroe, Trustee of the Ochsner Foundation, Mr. Albert Scheidt, the new hospital director, Dr. Dean H. Echols, director of the education and research program and Dr. Walter Trautman, a Fellow of the Foundation. The Benediction was by Reverend John S. Land. Mr. Monroe, who gave the main talk of the afternoon, pointed out the difficulties that were encountered in organizing this new hospital. He spoke, as always, most felicitously and most entertainingly. He said that the hospital, which had been opened on January 20, 1947 with the admission of three patients, now had 166 beds occupied and that they were prepared to open additional units.

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L. S. U. SCHOOL OF MEDICINE

Doctor Edgar Hull, professor of Medicine, was a guest of the L. S. U. Medical Alumni at Shreveport on April 2 and 3. While there he met with the alumni group on several occasions, gave three lectures on medical topics and conducted ward rounds in Shreveport Hospital and a clinical pathological conference.

Doctor Hull attended the meetings of the American College of Physicians at Chicago on April 28-May 2. He is Governor of the College for Louisiana.

Doctors Charles M. Goss, professor, Ralph N. Baillif, assistant professor and George W. Hamlett, assistant professor of anatomy, attended and presented papers at the meeting of the American Association of Anatomists at Montreal, Canada, on April 3 and 5.

Doctor G. M. McCoy, professor and head of the Department of Public Health attended the following meetings: April 12, 1947, American Epidemiological Society, Boston, Mass.; May 6-7, Association of American Physicians, Atlantic City, N. J.

Doctor Alfred B. Longacre, clinical instructor in Surgery, has received a grant of $3,000.00 from Eli Lilly Research Fund for the purpose of conducting studies in nitrogen balance following the administration of protein hydrolysates.

Doctor Ralph F. Brauer, recently appointed assistant professor of pharmacology at the L. S. U. School of Medicine, and at present a member of the faculty of the Harvard Medical School, has received a grant of $5,000.00 from the National Institute of Health for research in the effect of liver injury upon liver and plasma proteins. Doctor Brauer will join the faculty at New Orleans and begin his studies on July 1.

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THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The New Orleans Graduate Medical Assembly at a meeting held April 25, elected the following members for office for the year 1947-1948: Dr. Waldemar R. Metz, President-elect; Dr. Dean H. Echols, First Vice-President; Dr. Neal Owens, Second Vice-President; Dr. Maurice Lescale, Third Vice-President; Dr. Max M. Green, Secretary; Dr. Edwin H. Lawson, Treasurer; Dr. Edgar Hull, Director of Program; Dr. C. J. Tripoli, Asst. Director of Program; Dr. C. Gordon Johnson, Asst. Director of Program. Executive Committee: Dr. Hugh T. Beacham (two year term); Dr. John T. Sanders (two year term).

The following officers elected in 1946 for the coming year are as follows: Dr. Joseph S. D'Antoni, President. Executive Committee: Dr. William H. Gillentine, Retiring President; Dr. Lucien A. Fortier, Dr. Howard R. Mahorner.

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THE CENTRAL ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS

The Central Association of Obstetricians and Gynecologists, whose membership includes physicians from your State, offers two $100.00 each annual prize awards to any accredited physician, research worker, or medical student within the confines of the Central Association. Your State is within the confines of the Association. One award is for the best investigative work and the other for the best clinical work in the field of Obstetrics and/or Gynecology. Papers submitted for either of these awards must be in the hands of the Secretary not later than August 15, 1947.

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NEWS ITEMS

The ninety-fourth annual meeting of the New Orleans Academy of Sciences was held April 25-26. Dr. J. N. Ané and Dr. Mayerson, of Tulane, appeared on the program.

Dr. Michael E. DeBakey, Associate Professor of Surgery at Tulane University Medical School, was one of four additional physicians who were recently appointed honorary consultants to the Army Medical Library.

At a recent meeting of the Tulane Chapter of the American Association of University Professors Frank Lais, Jr., Executive Director of the Louisiana Physicians Service, Inc., addressed the members of the organization on the subject of surgical insurance.

The American Congress on Physical Medicine will hold its twenty-fifth annual scientific and clinical session September 2-6 at the Hotel Radisson, Minneapolis.

Dr. Bernardo Alberto Houssay, of Buenos Aires, Argentina, renowned as the world's greatest living physiologist, is to be the recipient of the first annual Research Award of the American Pharmaceutical Manufacturers' Association. Dr. Houssay is coming to this country at the APMA's invitation to receive the award in person. Dr. Anton J. Carlson, Emeritus Professor of Physiology at the University of Chicago who is known as the dean of American physiologists, will deliver the presentation address.

The National Gastroenterological Association will hold its twelfth Annual Convention and Scientific Sessions at the Hotel Chelsea in Atlantic City, N. J., on June 4-6, 1947, affording those interested in attending the centennial celebration of the American Medical Association and the meeting of the National Gastroenterological Association a chance to be present at both.

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POLIO PREPAREDNESS MEETING

The Louisiana State Department of Health, the National Foundation for Infantile Paralysis and the Orleans Chapter of the National Foundation jointly and holding their Polio Preparedness Meeting at the St. Charles Hotel in New Orleans, Friday, May 30, and Saturday, May 31.

The program provides for a general session to begin at 2:00 p. m., Friday, May 30 in the Jackson Room of the St. Charles, followed by a dinner meeting that evening at 7:30 p. m., in the Claiborne Room. Saturday morning, May 31, beginning at 9:30 a. m., there will be a professional session in the Beauregard Room. This meeting is exclusively for those who are engaged or interested in the medical care of infantile paralysis victims. Doctors, nurses, hospital people, medical social workers, physical therapists, are invited to attend the professional session.

Appearing on the program at the opening session will be Dr. W. L. Treuting, Dr. O. P. Daly and Dr. Kenneth Landauer, Director of Medical Care of the National Foundation for Infantile Paralysis. At the banquet in the evening, Mr. R. McLean Jeter will respond to the Welcome Address by Mayor Morrison. The two speakers at the banquet are Dr. G. W. McCoy, of the Louisiana State University Medical School and Dr. Clarence H. Webb, of Shreveport, Louisiana Chairman of the American Academy of Pediatrics. At the professional sessions Dr. Gilbert C. Anderson will preside and papers will be read by Dr. Myron Wegman, Professor of Pediatrics, Louisiana State University Medical School, Dr. Ralph Platou, Professor of Pediatrics at Tulane, and Dr. Guy A. Caldwell, Professor of Orthopedics at Tulane.

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NATIONAL ADVISORY HEALTH COUNCIL

Medical Research grants amounting to more than two million dollars have been recommended for the approval of Dr. Thomas Parran, Surgeon General, United States Public Health Service, by the National Advisory Health Council. Grants are contingent upon appropriations by the Congress for the fiscal year 1948 which begins July 1, 1947.

Of the 193 grants that supplement existing funds of universities and other research institutions, the largest, $105,800, was recommended for a study of syphilis by the Pan American Sanitary Bureau. Sums of $52,454 and $46,000 respectively, were slated for the study of malaria by the University of Chicago and Christ Hospital, Cincinnati, Ohio. Six other grants were above a $30,000 level.

Research studies involved cover a diverse number of subjects including tropical diseases, biochemistry and nutrition, cardiovascular diseases, dental research, gerontology, hematology, pathology, physiology, surgery, antiobiotics, tuberculosis, bacteriology, pharmacology, radiobiology, metabolism and endocrinology, sanitation, virus and rickettsial infections, and public health methods. The use of grants in aid in pursuing these studies implies no degree of Federal control. The investigator may work with full independence and autonomy, submitting only a brief concise report of scientific progress annually.

An additional 55 grants were favorably considered but await modification of amount or further study by the Research Grants Division of the National Institute of Health before final action is taken.

The National Advisory Health Council is established by Congress and aids the Surgeon General in carrying out the research programs of the Public Health Service. It consists of 14 members, 10 of whom are experts, not otherwise employed by the government. They are: Dr. Edwin B. Fred, President, University of Wisconsin; Dr. Karl F. Meyer, Director, Hooper Foundation for Medical Research, University of California; A. Baird Hastings, Department of Biological Chemistry, Harvard University; John H. Musser, Professor of Medicine, Tulane University; Henry F. Vaughan, Dean, School of Public Health, University of Michigan; Harry S. Mustard, Professor of Public Health Practice, College of Physicians and Surgeons, New York City; Gordon H. Fair, Professor of Engineering, Harvard University; Carl S. Marvel, Professor of Organic Chemistry, University of Illinois; William C. Rose, Professor of Biochemistry, University of Illinois; and the most recent appointee, Dr. George Baehr, Clinical Professor of Medicine, Columbia University.

Four ex-officio members include the Director, National Institute of Health, Dr. Rolla E. Dyer; the Chief, Bureau of Medicine and Surgery, Navy Department, Captain O. L. Burton, USN; the Chief, Preventive Medical Services, Army Service Forces, Colonel Karl R. Lundeberg, USA; the Assistant Chief, Bureau of Animal Industry, Department of Agriculture, Dr. Harry W. Schoening.

ATTENTION NAVAL RESERVE MEDICAL OFFICERS!

The attention of Reserve Medical Officers is invited to the opportunity to return to *active duty* at one of the major naval air stations of the Naval Air Reserve Training Command or at one of the Naval Air Reserve Training Units (NARTUs) listed below:

NAS Atlanta, Ga.
NAS Columbus, Ohio
NAS Denver, Colo.
NAS Dallas, Texas
NAS Glenview, Ill.
NAS Crosse Ile, Mich.
NAS Los Alamitos, Calif.
NAS Memphis, Tenn.
NAS Miami, Fla.
NAS Minneapolis, Minn.
NAS New Orleans, La.
NAS New York, New York
NAS Oakland, Calif.
NAS Olathe, Kans.
NAS Squantum, Mass.
NAS St. Louis, Mo.
NAS Willow Grove, Pa.
NARTU, NAS Anacostia, D. C.
NARTU, NAS Jacksonville, Fla.
NARTU, NAS Norfolk, Va.
NARTU, NAS Seattle, Wash.

Reserve Medical Officers who are interested in *active duty* at one of the stations or units listed above should initiate letters to the Bureau of Naval Personnel, via Chief of Naval Air Training, Naval Air Station, Glenview, Ill., and BuMed, listing three or four stations at which duty is desired in order of preference. Personnel are desired in rank of Commander and below in the Medical Corps. However, Captains may apply for duty in their rank, requesting waiver.

Officers qualifying for the above billets will not be subject to transfer and may terminate the tour of duty at own request. Flight Surgeons are assured of orders to duty involving flying. Government quarters are available at several of the major naval air stations. *Here is a chance for a reserve officer awaiting residency or fellowship in a civilian hospital to obtain a full time paying job in the meantime.*

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HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week closing March 15 there occurred in the City of New Orleans 156 deaths which were divided 113 white, 43 colored, with 11 of these deaths in children under one year of age. The three year median for the corresponding week shows 126 deaths so that this was not a particularly happy week for the citizens of New Orleans. The following week there was a marked improvement, the number of deaths reported being

127; 79 of these were in the white population and 48 in the colored with only seven of the total being in infants. There was a very large jump the following week which closed March 29. The number of deaths ascended to the high figure of 169. The racial distribution of these deaths was as follows: white 106, colored 63, with 18 infant deaths.

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INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week ending March 8 showed cancer leading all other reportable diseases with a total of 153 cases. Following cancer, numerically, came unclassified pneumonia with 128 cases reported, 59 of measles, 57 of pulmonary tuberculosis, 34 of pneumonoccic pneumonia, 18 of influenza, 13 of amebiasis and 10 of chickenpox. There were no other diseases recorded in figures greater than ten. It should be noted, however, that four cases of poliomyelitis were reported. The following week which terminated March 15 saw unclassified pneumonia the first of all other reportable diseases with 101 cases. Other diseases occurring in numbers greater than 10 included 56 of measles, 42 of pneumococcic pneumonia, 35 of pulmonary tuberculosis, 19 of cancer, 18 of chickenpox, 13 of diphtheria and 12 of scarlet fever. This week there were six cases of poliomyelitis listed, scattered in various parishes throughout the state. For the first time in many months a case of pellagra was reported. The week which closed March 22 mumps led the reported diseases listed with 169 instances, followed by cancer with 119 cases, influenza with 85, chickenpox 66 and measles 42. The very high figure of unclassified pneumonia, 281, for the previous four weeks was remarkable as there were only five cases listed this week. Only one case of poliomyelitis appeared in the state this particular week. Influenza was apparently rampant the last part of March because 315 cases were reported for the week which closed March 29. Other diseases that were reported included 117 cases of measles, 76 of chickenpox, 68 of pulmonary tuberculosis, 40 of cancer, 36 of amebiasis, 14 of unclassified pneumonia and 11 of mumps. Again there was only one case of poliomyelitis reported.

Month Ending February 28, 1947

	Total This Month	Total Previous Months	Total to Date 1947
Chancroid	57	58	115
Gonorrhea	1214	1284	2498
Granuloma Inguinale	9	9	18
Lymphopathia Venereum	10	9	19
Syphilis	844	966	1810

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JANE TODD CRAWFORD STUDENT LOAN FUND IN GYNECOLOGY

The Woman's Auxiliary to the Southern Medical Association has established a loan fund for young Southern physicians wishing to pursue the study of gynecology. The requirements are simple: he or she must not be over 32, of good character, and showing evidence of being capable of absorbing adequate knowledge.

Inquiries may be addressed to Mrs. J. Ullman Reaves, 1862 Government Street, Mobile 18, Alabama.

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AMERICAN PSYCHIATRIC ASSOCIATION

Child Psychiatry, Convulsive Disorders, Psychiatry and the Law, and the U.S.P.H.S. Program in Mental Health are among the topics to be reviewed at the annual meeting of the American Psychiatric Association to be held May 19-23 in New York City. The discussion will be led by such men as Dr. G. Brock Chisholm, Dr. Paul R. Hawley and Dr. William C. Menninger.

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LOUISIANA PHYSICIANS SERVICE ENJOYS RAPID GROWTH

The Louisiana Physicians Service, the Blue Shield Plan for surgical and obstetrical services, has now grown where it has more than 12,500 individuals covered by its program. The plan has had an enrollment increase since January 1, of 109 per cent through March 31 of this year.

The Blue Shield Plan as offered by Louisiana Physicians Service is the only plan sponsored in Louisiana by the doctors and is sponsored and approved by the Louisiana State Medical Society. The plan now has an actual net premium value of approximately $90,000. The plan started just about five months ago.

The "Blue Shield" Plan in Louisiana has been granted the Seal of Acceptance of the Council on Medical Service of the American Medical Association, and is one of the eighteen selected plans in the United States to enjoy full membership in Associated Medical Care Plans. Associated Medical Care Plans is a national organization for prepayment medical care plans.

The doctors' plan provides for the prepayment of surgical and obstetrical care and includes treatment of fracture and dislocations along with certain related benefits such as diagnostic x-ray, anesthesia, and laboratory services. By the payment of a small fee the employed individual may prepay the cost of his doctor's bill and through this method of cooperative participation eliminate the hazard of the catastrophic doctor's bill. The Blue Shield Plan offers, through the participating physician, complete surgical and obstetrical coverage to individuals whose annual income is less than $2,000 per year and to the family group whose aggregate annual income is less than $3,000 per year. This means that approximately 780 physicians throughout Louisiana have voluntarily agreed to perform complete surgical and obstetrical services to individuals who come to them for service and whose income does not exceed the amount stated.

For the individual whose income is in excess of the amount stated the plan acts as an indemnity toward the doctor's bill. The plan is available to all persons irrespective of their annual income.

The subscriber has complete freedom of choice of his physician and may use any doctor anywhere in the United States and will be paid the amount for surgical procedure rendered as provided for in the schedule of the benefits. However, persons using a participating physician and whose income is below the amount stated are guaranteed complete service at no extra cost.

The plan is operated on a state-wide basis and at this time has established offices in the cities of Baton Rouge, Alexandria, Shreveport and Monroe. The home office of the company is in New Orleans.

Louisiana Physicians Service will have a booth at the annual meeting of the Louisiana State Medical Society and all doctors interested in obtaining additional information relative to the operation of the "Blue Shield" are invited to inquire at the booth where an individual explanation will be made. Additional literature on the plan may be obtained at the booth, or by writing to the home office of the company, Room 103, 1430 Tulane Avenue, New Orleans.

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DOMINICK A. PALMISANO

1888-1947

Dr. Dominick A. Palmisano died Monday, April 21 at Hotel Dieu in New Orleans. Dr. Palmisano was 56 years of age, graduated from Tulane in 1913 and was in general practice in New Orleans until 1932. From that time on he specialized in eye, nose and throat work. He was a staff member of the Eye, Ear, Nose and Throat Hospital, Hotel Dieu, Woman's Dispensary and Charity Hospital, as well as a member of parish, state and national medical societies.

Dr. Palmisano's death will be a real loss to his numerous friends and patients.

DAVID ADIGER

1889-1947

Dr. David Adiger died suddenly in New Orleans, at the age of 58 years, on April 2, 1947. Dr. Adiger was born in Poland in 1889, his family settling in St. Francisville a short time after his birth. He graduated from Tulane Medical School in 1913. After serving a two year internship at Charity Hospital he was house surgeon for a year.

Dr. Adiger was a member of the parish and state medical societies and of the American Medical Association and had been since 1915. He served two years in World War I and on his return began to specialize in gynecology and obstetrics. Dr. Adiger was a man of outstanding character. He was deeply beloved because of a warm-hearted personality and high ideals.

PHYSICIAN WANTED

Doctor in rural community is retiring from practice in June, 1947 and would like to have doctor take over his practice. The town is on a paved highway and there is a radius of at least twelve miles in which to do general practice. An annual gross income of $12,000.00 is estimated. Well equipped office with x-ray, diathermy, ultra violet light, infra red light, and other appliances. Hospital facilities six miles from office. For further information communicate with office of New Orleans Medical and Surgical Journal.

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WOMAN'S AUXILIARY

I am very grateful to Mrs. Milton Honigman for all the wonderful publicity furnished me from Rapides Parish Auxiliary. The members did a fine job with their publicity, for Mrs. Rex G. Fortenberry's lecture. In addition to the usual poster and printed advertisements they had radio publicity over Station KALB, with three announcements over this station. Rapides Auxiliary is taking a very active part in Cancer Control and will hold a special meeting in cooperation with the American Society for Cancer Control on February 28, with Mrs. H. C. Graham as chairman.

One of the outstanding functions of the year was the first fall meeting honoring Mrs. Arthur Long of Baton Rouge with a luncheon, in the Bentley Room of the Bentley Hotel with a large crowd in attendance.

During the Christmas holidays, a very festive event for doctors and their wives was enjoyed in the form of a dinner party at "Homewood," the palatial home of Mrs. Clarence Pierson, one of the cities most charming hostesses.

This is only some of the fine work Rapides Parish Auxiliary is doing. The results have been very gratifying.

Ouachita Parish has done some fine work and is also a very active and outstanding Auxiliary. They too worked long and untiringly on publicity preceding the lecture of Mrs. Rex Fortenberry, and the response was indeed gratifying.

Ouachita Auxiliary feels fortunate in having as a member Mrs. A. D. Tisdale who is a member of

State Presidents of Federated Clubs and to have her give her report on the New York Herald Tribune Open Forum which she attended in New York. She gave special emphasis on cancer control and research in the field of atomic energy in combating cancer as reported at the Open Forum.

The Hygeia Chairman, Mrs. J. E. McConnell, reported more subscriptions sold than ever before in that Auxiliary.

At a Christmas luncheon, Dr. A. L. Anderson, Superintendent of the G. B. Cooley Sanitarium, spoke on anti-tuberculosis. He set forth a five year program planned for combating tuberculosis in Ouachita Parish and inspired the Auxiliary to aid the program in every way possible.

Ouachita Auxiliary honored their doctors on Doctor's Day with a specially planned dinner party.

You can see from this fine report that Ouachita Parish has had a full program representing plenty of time, effort and hard work.

Another very wonderful report and one that we will all enjoy reading is that of Caddo Parish Auxiliary. In October this Auxiliary was honored by a visit from the State President, Mrs. Arthur Long, of Baton Rouge. A beautiful tea was given for Mrs. Long and the happy occasion was one to be long remembered by all attending. Mrs. Long was also named honor guest at a luncheon given by officers and board members of the Caddo Parish Auxiliary.

At the November meeting a very interesting report was given by Mrs. C. R. Gowen, Chairman of Cancer Control, on the district meeting of the American Cancer Control Society. The Auxiliary voted to sponsor the making of surgical dressings for terminal cancer cases. Dr. Lloyd W. Rowland, Director of the Louisiana Society for Mental Health, gave a most interesting and instructive discussion on the subject.

At the December meeting, Dr. M. E. Dodd, guest speaker, spoke of "Life Among the Maoris of New Zealand," stressing points of medical and social interest. The Auxiliary members brought toys which were distributed to the children of the Charity Hospital in Shreveport at a Christmas party.

Several of the members were quite active in the Christmas seal drive. Many members have taken an active part in the work of the Cancer Society and have sponsored a surgical dressing center.

This is only a few of the many worthwhile things accomplished by Caddo Parish Auxiliary. I wish that space permitted more be printed.

MRS. W. C. WIGINTON,
Chairman, Press and Publicity.

IN MEMORIAM

Died August 9, 1946: Mrs. C. P. Rutledge—Past President.

Died December 4, 1946: Mrs. F. A. Buvens.

BOOK REVIEWS

Insect Microbiology: By Edward A. Steinhaus. Ithaca, N. Y., Comstock Publishing Company, Inc., 1946. Pp. 763. Price $7.75.

Dr. Steinhaus who is an authority in the field has here summarized current knowledge concerning the associations of microbes with insects and ticks. The book is particularly noteworthy for its comprehensiveness, since it treats bacteria, rickettsiae, viruses, yeasts, fungi, spirochetes and protozoa. For example, nearly 100 pages are devoted to a discussion of the rickettsiae while over 70 pages of the text deal with intracellular bacterium-like or rickettsial-like symbiotes. There is an extensive bibliography of pertinent literature at the end of the book, together with subject and author indexes. Microphotographic reproductions and diagrams are numerous and for the greatest part, exceedingly good. The book can be highly recommended as a reference source to all students of medicine as well as those interested in veterinary or plant infectious diseases and entomology.

M. F. Shaffer, Ph.D.

Music in Hospitals: By Willem van de Wall, New York, Russell Sage Foundation, 1946. Pp. 86.

Not many books of this type appear on the market. The value of this work, however, lies not in its uniqueness but in the evident knowledge of the author of the field with which he deals in the course of the work.

It has long been axiomatic that music is, in itself and in its appreciation, an excellent therapy for human ills, particularly of the emotional type. And in recent years some effort has been made to make use of this age-old remedy in the rehabilitation of those whose emotional unbalance has made them ill. However, few if any authors have so worked out a plan as has Mr. van de Wall.

The work is most certainly worthy of commendation. Of particular value are his studies of the effect of music in normal living, the nature of individual responses to music, and, most especially, the chapter in which he details the technic of actual hospital musical programs. There has been much confusion of thought and practice in the latter field. Musicians have been willing to render their services, but so many haphazard acceptances of their attempts have tended, in many cases to frustrate the effectiveness of musical therapy, rather than to permit the application of its full value. A definite, graduated program—or at least the suggestion of a method of organizing one—has long been needed. It is most gratifying to find the author of this work offering such a clear delineation of the most effective methods of procedure.

Music in Hospitals is a small work but mighty—for it fills a long-felt need. The author modestly refers to it as: "a collection of thoughts and suggestions based on practical experience and offered for purposes of information and stimulation." It is far more than that. May it fulfill its purpose, so well presented by the author, of developing interest in music as a therapy for healthier emotional living.

B. J. Cunningham, C. M.

Henry Sewall, Physiologist and Physician: By Gerald B. Webb and Desmond Powell. Baltimore, The Johns Hopkins Press. 1946. Pp. 189. Price $2.75.

Henry Sewall started his long scientific career in 1876 as an assistant to Henry Newell Martin, the first professor of physiology at Johns Hopkins University. After three years under Martin he spent several years abroad, studying with Ludwig in Leipzig, Kuhne in Heidelburg, and Sir Michael Foster in London. In 1882 he was called to the University of Michigan to organize the Department of Physiology in its medical school.

The rest of the story of his life until his death in 1936 is a record of remarkable achievement in the face of heroic struggle against tuberculosis, which Sewall developed soon after going to Ann Arbor. In between bouts of illness he found time to teach, to make many friends, and to do experimental work. When he finally realized that he could not endure the climate in Michigan, he gave up his post in physiology, left Ann Arbor, and courageously entered the practice of medicine. After a year as resident physician with Trudeau at Saranac, Sewall migrated to Denver, where he taught physiology and medicine in the University of Colorado Medical School for approximately thirty years, while carrying on an active medical practice.

While Sewall made some valuable contributions to physiology during his years in Michigan, the work for which he is most famous is the proof of the efficacy of immunization to snake venom. These experiments were published in 1887, and as the authors point out, "Sewall did more than to establish a fact; he recognized its implications and pointed them out to future investigators."

As a physician, Sewall devoted himself to the problems of his community and of medicine at large. He pioneered in public health measures, wrote vigorously against the anti-vivisectionists, and became a leader in the field of tuberculosis. His services were much in demand as a consultant and as a leading citizen.

The authors have told the story of this remarkable man in an interesting and sympathetic fashion. Sewall worked through an exciting, pioneer period in physiology and medicine. In telling the story of Sewall's life and achievement, the authors have, of necessity, recounted much of the progress of medical investigation and thought of the early part of the century.

H. S. Mayerson, Ph. D.

Urgent Surgery, Volume I: Ed. by Julius L. Spivack, M. D., LL. D. Springfield, Charles C. Thomas, 1946. Pp. 714. 244 fig., 572 illus., 14 in color. Price $10.

This is a difficult book to evaluate fairly. For one thing, this is volume I, but nowhere in the preface or in the publisher's notice on the cover is there any intimation of the scope of the proposed work, how many other volumes there will be, or what subjects they will treat. This volume, which is the work of seven contributors in addition to the editor, contains eighteen chapters, thirteen of which deal with intra-abdominal conditions. The other five chapters are concerned, respectively, with intravenous infusion and blood transfusion; shock, hemorrhage, septicemia and gas gangrene; anesthesia; electrosurgery; and laparotomy. The inclusion of gas gangrene, the discussion of which is chiefly concerned with its occurrence in the extremities, in this particular volume does not seem entirely logical, nor does a chapter on electrosurgery seem indicated in a volume dealing with urgent surgery.

The editor's definition of urgent surgery seems itself somewhat confusing. Emergency surgery, he points out in the preface, some years ago was limited to accidents, principally traumatic, and to tracheal obstruction by foreign bodies. The development of diagnostic facilities (should not the development of surgical technics and of anesthesia also be included?) led to surgery for ruptured viscera and for intestinal obstruction. Today, the elements of time and space have been overcome by rapid transportation. Even in rural districts, well equipped hospitals staffed by competent surgeons are within comparatively easy reach (a generalization with which some observers would quarrel), and on that basis alone the term emergency surgery, the editor believes, can justifiably be replaced by the term urgent surgery. Emergency surgery implied the necessity for intervention within minutes or hours. Urgent surgery is concerned with conditions in which operation becomes imperative within a few days if life and health are to be preserved. The notice on the cover states that the book deals with conditions requiring immediate or almost immediate intervention, and, in particular, with conditions in which operation becomes imperative within a day, or at the most within two days, if life and health are to be preserved.

The reasoning is difficult to follow, and more difficult to correlate with the text. Surely the availability of transportation and of a good surgeon has nothing to do with the concept, which still prevails, that acute appendicitis, for instance, or intestinal obstruction, is a condition in which the sooner operation is performed the better, and in which the mortality increases with each hour of delay. Carcinoma of the large bowel, like all malignancy, is urgent in the sense that the patient will certainly die without radical operation, but abdominoperineal resection is not usually conceived of as urgent surgery. Furthermore, if carcinoma of the large bowel is regarded as an urgent condition, it would seem reasonable to include in the same category carcinoma of the stomach, which is more frequent, but which is not discussed at all except in a section devoted to perforation of gastric malignancy. Six pages are devoted to volvulus of the stomach, of which less than a hundred cases have been reported, but less than a page to volvulus of the colon, which is many times more frequent.

In general, there can be little quarrel with the methods of therapy proposed for the conditions discussed. There is proper emphasis on typical cases of acute appendicitis, but the discussion of what happens clinically twenty-four and forty-eight hours after the onset of the disease is much too leisurely; acute appendicitis can be far advanced at the end of the second day. Not sufficient emphasis is placed upon the serious potentialities of the obstructive type of acute appendicitis, nor would there be universal agreement with the statement that statistics prove the value of abstinence from surgery in peritonitis following rupture of the appendix. Exception might well be taken, too, to the conservative therapy of appendiceal abscess in childhood. Far more emphasis should be put upon the dangers of primary resection in the face of malignant obstruction of the colon, which carries a prohibitive mortality.

The bibliographies are uneven. There are many recent references, but many others which could profitably be replaced by later ones; some of them would invalidate the earlier references. In the chapter on transfusion, for instance, the most recent reference is to a text published in 1942, which naturally takes no account of the enormous military experience with transfusion accumulated after that date. The same comment is applicable to the references on shock. There are two references to Moon's somewhat embattled concepts, but no reference at all to the experiences of World War II, which overturned a good many former theories.

In many instances references in the text are not listed in the chapter bibliographies. Some names are spelled incorrectly. Exact statistics are frequently cited without their source. Incidentally, the background statistics for acute appendicitis are for the years 1935 and 1936. Bureau of Census statistics are available in detail through 1945, some for 1946 are now available, and comparative statistics are also available as late as 1944. Acute appendicitis is still a very serious disease, but the last three or four years have shown an enormous improvement, and quotation of ten-year-old figures naturally does not reflect it.

In spite of these strictures the book has numerous good points. Cole's chapter on the pancreas, for instance, is very well done and sticks close to the ordinary concept of emergency (urgent) conditions. Paine's chapter on intestinal obstruction is equally good. The illustrations, done under the direction of Tom Jones, are generally clear and helpful.

Frederick Fitzherbert Boyce, M. D.

Gynecological and Obstetrical Pathology, with Clinical and Endocrine Relations: By Emil Novak, A. B., M. D., D. Sc. (Hon. Dublin), F. A. C. S. 2d ed. Philadelphia, W. B. Saunders Co., 1947. Pp. 570. 542 illus., 15 in color. Price, $7.50.

The second edition of Gynecological and Obstetrical Pathology by Dr. Emil Novak is more than welcome. The first edition filled a much needed place in the library of the clinician, pathologist and student of gynecology and obstetrics, and this edition brings the subject up to date. The second edition follows the same format as the previous, beginning with a discussion of the endocrinology of the menstrual cycle and pregnancy and following with the discussion of the various organs of the female genital tract, including vulva, vagina, cervix, endometrium, uterus, fallopian tubes, uterine ligaments and ovaries with brief review of the embryology and histology of each, as well as the pathology.

The chapters devoted to the tumors of the ovary are of particular interest and represent as authoritative a compendium on the subject as it is possible to obtain in a single text, particularly when one considers that the particular subject matter is a favorite of Dr. Novak, and one in which he is probably "the" authority today.

The remaining chapters dealing with ectopic pregnancy, endometrial implantation and placentation, hydatidiform mole, chorio-epithelioma-malignum and abnormalities and diseases of the placenta and appendages are no less valuable in content.

The addition of many new photographs, microscopic and gross, including several color plates, with close correlation to text, enhance the value of this text as an instrument of ready reference as well as an aid to teaching.

Last but not least, Dr. Novak's refreshing style makes the reading enjoyable as well as profitable. Chapter references are of inestimable aid to those who wish to pursue more deeply into selected topics. This text should be regarded as required reading, for everyone interested in the diseases of women and should be readily available for constant and repeated reference.

J. H. Collins, M. D.

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Modern Management in Clinical Medicine: By F. Kenneth Albrecht, M. D. Baltimore, The Williams and Wilkins Company. 1946. Pp. 1238 illus. Price $10.

Careful study of this useful volume convinces one that it is the contribution of a clinical pragmatist whose experiences in ward and clinic have been both extensive and fruitful. Dr. Albrecht's avowed effort to present comprehensive and modern material upon the management of clinical entities in readily available form has been successful. Especially valuable is the exhaustive consideration of semiological approaches to the adequate study of clinical cases. Excellent illustrations, many in full color, contribute much to clarity and forcefulness of presentation. The prime feature of the contribution is that specific and detailed recommendations are made in considering the therapy of each entity; the fact that trade names of therapeutic agents are used enhances the value of the material.

Chapters on the management of common dermatoses, venereal diseases and geriatric problems are especially good.

In the discussion of the management of diabetic acidosis the vehemence of one statement: "*The second most serious error one can commit is to administer glucose*" may not win complete agreement from many clinicians of wide experience.

Since it has a well-organized index, a pleasing format and presents valuable material in readily available form the volume "wears well" in daily use. It should have wide appeal, particularly to busy practitioners of general medicine.

Edwin S. Kagy, M. D.

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A Handbook of Commonly Used Drugs: By Michel Pijoan, B. A., M. D. & Clark H. Yaeger, M. D., Dr. P. H. Springfield, Charles C. Thomas. 1947. Pp. 198. Price $3.75.

As stated in the preface this book represents the accumulation of notes dealing with drugs, preparations and technics which have been found useful in the actual experience of the authors. Among the groups of drugs considered are the analgesics and hypnotics, central nervous system stimulants, autonomic drugs, cardiovascular drugs, diuretics, oxytocic agents, hormones, vitamins, hematologic agents, metallic compounds, general antiseptics, sulfonamides, antibiotics, etc. Since the scope approaches that of most texts on pharmacology the discussion in each case is necessarily synoptic in a volume of this size. Furthermore the extent of treatment is undoubtedly influenced by the personal interests and experience of the authors. For example, the general anesthetics are omitted and ergonovine is dismissed with two short sentences while approximately one quarter of the book concerns the treatment of tropical diseases. Therapeutic applications rather than mechanisms of action are usually stressed. In an appendix are a number of useful prescriptions and other miscellaneous material. Although this book contains much useful information the physician is advised to examine it before purchasing it in order to assure himself that it will satisfy his needs in this field.

Ralph G. Smith, M. D.

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Postgraduate Obstetrics: By Wm. F. Mengert, M. D., New York, Paul B. Hober, Inc., 1947. Pp. 392. Price $5.00.

Written primarily for the general practitioner, this book covers adequately the usual obstetric problems encountered and places less emphasis on rarer complications, theory being kept to a minimum.

The diagnosis of pregnancy, prenatal care, minor ailments of pregnancy, related disease complications and unrelated intercurrent diseases, abortion and premature labor, hemorrhages, cesarian section, normal conduct of labor, analgesia, forceps, prolonged labor, the normal and abnormal puerperium, care of the newborn, sterility, laboratory procedures and nursing technics are all covered in an adequate manner. The indications and contraindications relative to cesarean section are excellent. General principles are more emphasized with less attempt at great detail, in most sections of the book, in a clear, concise, readable fashion. This is one of the stated purposes of the author and a valuable feature of his book, in his avoidance of theory and controversy.

Some might question a few recommendations as to the termination of pregnancy in the second trimester by rupture of the membranes and occiput posterior might have been given more emphasis, but by and large this is an excellent conservative, thoroughly modern book on obstetrics primarily written for the man that delivers most of America's babies—the family doctor.

Calvin M. Johnson, M. D.

Compleat Pediatrician: By W. C. Davison, M. A., D. Sc., M. D. Duke University Press, Durham, N. C., 1946. Pp. 256. Price $3.75.

The Compleat Pediatrician is an old book brought up to date. I have always had a copy of this in my library and think it is the most concise, detailed reference a pediatrician can find. The man doing general work will find help for any of his pediatric problems here.

Suzanne Schaefer, M. D.

PUBLICATIONS RECEIVED

Froben Press, New York: If You Need an Operation by Richard A. Leonardo, M. D., Ch. M., F. I. C. S.

Lea & Febiger, Philadelphia: The Head, Neck and Trunk by Daniel P. Quiring, Ph. D.; Therapeutic Exercise by F. H. Ewerhardt, M. D. and Gertrude F. Riddle, B. S., R. N., R. P. T.

Mack Publishing Company, Easton, Pa.: The Pharmacopoeia of the United States of America, Official Copy No. B 963.

Williams & Wilkins Company, Baltimore: Obstetrical Practice by Alfred C. Beck, M. D.

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AN EVALUATION OF ESTROGEN THERAPY*

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AND
S. LEON ISRAEL, M.D.†
PHILADELPHIA, PA.

It is impossible to consider in a single paper our present knowledge of the hormones of the ovary, a field of research and therapy which encompasses an enormous section of medical knowledge and theory. The original concept of the ovary as a single endocrine gland which could produce symptoms by means of quantitative variations of its secretions was a simple explanation of a now multi-faceted fact. The advances of the last two decades have enlarged the problem by making available clinically useful preparations of the two hormones of the ovary, estrogen and progestin. This review deals with estrogen—the hormone of growth and preparation—the more widely employed of the two.

When estrogen first became available for clinical use, there was an understandable enthusiasm of its application but the true indications for its use have contracted considerably following the earlier empirical trials. Because of the complexity of the problems for which estrogen is used and because of its multiple commercial forms, much confusion still exists, particularly concerning dosage and the avoidance of undesirable side-actions. If one depends upon the beautifully embossed brochures which exort the practitioner with every mail, one falls, unhappily, into the security of the illusion that there are neither contraindications nor untoward side-effects in the use of estrogen.

THERAPEUTIC PRINCIPLES

Before discussing the specific abuses of estrogen therapy which the authors have recently encountered, it seems expedient to outline certain basic maxims which are essential for the proper use of any endocrine therapy.

1. *The Elimination of Organic Disease*: It is imperative that a complete survey of the patient be made to exclude the presence of any organic gynecologic disorder or constitutional ailment which would preclude endocrine therapy. This entails the use of all ordinary diagnostic procedures, including complete physical examination, blood count, urinalysis, serologic study, biologic pregnancy test, and determination of the basal metabolic rate. Such an effort at differential diagnosis is of special importance before the use of so potent a substance as estrogen.

2. *Individualization*: The knowledge that no two patients ever present the same problem in the same manner is of paramount importance. The value of individualization is clearly emphasized by the varying treatment of the same disorder at different age levels. Moreover, treatment must be directed not only to the disorder in question but the therapeutic regime must be so instituted that associated derangements are corrected. It is important to remember

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*Read before the New Orleans Graduate Medical Assembly, February 24-27, 1947.

†From the Department of Gynecology and Obstetrics, Graduate School of Medicine, University of Pennsylvania.

that a patient is not composed wholly of selected endocrine glands.

3. *Evaluation of the Pathologic Physiology*: A working knowledge of the normal menstrual cycle is of importance in the use of both estrogen and progestin. Despite the fact that most physicians have some understanding of the current conception of normal menstruation, and at the risk of being somewhat pedantic, the cardinal principles are reviewed briefly to lay emphasis on the necessity of appreciating the role which the ovarian hormones play in the cycle.

The cyclic ovarian changes are governed by the secretions of the anterior lobe of the hypophysis. The latter produces two gonadotropic hormones which are responsible for follicle maturation and corpus luteum formation, respectively. In turn, each of these two phases of the ovarian cycle controls a portion of the uterine cycle. The recurring changes in the endometrium are entirely dependent upon the hormones secreted by the ovarian follicles and the corpus luteum. The periodic, postmenstrual ripening of a group of ovarian primordial follicles is productive of estrogen, the hormone which effects the proliferative phase in the endometrium.

During the first half of the menstrual cycle, as a result of the ovarian follicular hormone, estrogen, the endometrium changes from a thin layer of cells practically devoid of glands to a tall, richly glandular tissue. At approximately the mid-interval of the cycle, one of the growing follicles matures, ovulation occurs, and the collapsed follicle is transformed within a few days into a corpus luteum. This change in the ovary is effected by the gonadotropic hormones of the anterior lobe of the hypophysis. The luteinizing gonadotropin supports the continued function of the corpus luteum. The lutein cells continue, as their forebears in the follicle did, to produce estrogen but, in addition, also secrete progestin, the characteristic hormone of the corpus luteum.

During the second half of the cycle, progestin causes the estrogen-primed endometrium to become a hyperemic, highly functional tissue richly endowed with nutritive substances required for the embedment and support of a fertilized ovum. If a fertilized ovum does not eventuate, the corpus luteum disintegrates and, almost immediately thereafter, the endometrium is dismantled by the process of menstruation.

Thus, it is clear that estrogen is the hormone of growth, vascularization, and preliminary preparation, whereas progestin is the principle which modifies and differentiates.

4. *Application of Basic Pharmacology*: The ovarian hormones belong to the steroid family, a group which also includes androgens and the adrenocortical hormones. The steroidal hormones are higher alcohols, possessing a common chemical denominator which relates them to such well-known substances as cholesterol, bile acids, certain opium alkaloids, stigmasterol, and various glucosides of digitalis. The ovarian steroids —estrogen and progestin—are chemically identified, their structural formulas are wellknown, they are available in pure crystalline form, and have been made synthetically. They are stable, do not require refrigeration, and will remain potent indefinitely. Inasmuch as they contain no protein, neither of them gives rise to allergic manifestations. They may be administered intramuscularly, orally, and by absorption through skin or mucous membrane. Each of them has the significant dual ability of acting on the genital end-organs and on the anterior lobe of the hypophysis. The latter characteristic is important to remember when treating a regularly menstruating woman because the rhythm of her cycle may be disturbed through depression in the production of hypophyseal gonadotropic hormones.

ESTROGEN USAGE

The physiologic effects of estrogen make it useful in two categories—either to achieve growth of all or part of the genital tract or to depress temporarily the function of the anterior lobe of the hypophysis. In the former instance, are included the treatment of atrophic vaginitis and the estrogen therapy of amenorrhea. The latter includes its uses for the suppression of lactation and the alleviation of the menopausal syndrome.

1. *Antrophic Vaginitis*: The predisposing cause of postmenopausal vaginitis is the extreme vulnerability of the atrophic vaginal mucosa, causes a diminution in the layers of the epithelium, a disappearance of the glycogen content of the cells, and a loss of the protective vaginal flora. Deprived of these natural barriers to infection, the vagina becomes fertile soil for bacterial growth. The atrophy and subsequent infection give rise to the symptoms of irritating leukorrhea, introital paresthesia, and dyspareunia.

Since the absence of estrogen is the basic cause for this disorder, it is logical to administer small doses of estrogen in order to restore the vaginal epithelium to its former height and state of resistance. The dosage of estrogen employed for this purpose need not be large. It may be effectively given orally in a dose of 0.5 mg. of stilbestrol or 0.625 mg. of estrone sulfate daily for several weeks. Another method of administering estrogen in such cases is the nightly introduction of a vaginal suppository containing 0.2 mg. of estrone. However prescribed, the estrogen should be discontinued in a month and repeated only if required for a recrudescence of symptoms.

2. *The Adjunctive Therapy of Amenorrhea*: Inasmuch as the causes of amenorrhea are numerous, a complete investigation of the constitutional background of each amenorrheal woman is essential to intelligent therapy. The treatment of amenorrhea with estrogen alone is notoriously unsatisfactory. Estrogen can produce only the proliferative phase of endometrial growth. Although uterine bleeding may result in amenorrheal women treated with estrogen alone, such bleeding is not menstruation.

The physiologic objective of estrogen therapy in amenorrhea is to combat uterine hypoplasia. If the uterine atrophy—perhaps, the sequel of a previously existing pituitary-ovarian deficiency—is the *sole* cause of the amenorrhea, good results may be obtained from estrogen treatment. In such women, the endometrium is rebuilt to a point of normal responsiveness and, in effect, the estrogen acts merely as the "starter" which sets the cycle in motion. Unfortunately, this applies to very few amenorrheal women.

An effective technic of employing estrogen in the treatment of amenorrhea is either its simultaneous combined, or its consecutive cycle administration with the corpus luteum hormone, progestin. Thus, in the former method, as employed for the usual type of secondary amenorrhea, the patient may be given both 2.5 mg. of estradiol benzoate and 12.5 mg. of progesterone intramuscularly on two successive days. With the latter, the cyclic method, one may administer 1.5 mg. of estradiol benzoate intramuscularly every third day for five doses and follow immediately with five daily injections of 10 mg. of progesterone. Such dual therapy, combined or cyclic, is generally repeated for three consecutive months, each succeeding course being started as the induced flow ceases.

When parenteral medication is to be avoided, one may employ orally administered estrogen and progestin. Such a schedule, for example, might be the use of 2 mg. of stilbestrol daily for 15 days, followed by 60 mg. of pregneninolone, the oral progestin, daily for five days.

It must be remembered that neither estrogen alone nor estrogen with progestin gives *permanent* results in amenorrhea unless other measures are taken simultaneously to restore the general health of the patient.

3. *The Prevention of Puerperal Breast Engorgement and the Suppression of Lactation*: One of the most effective uses of estrogen, whether natural or synthetic, is its ability to prevent breast engorgement in instances of postpartum inhibition of lactation. When the administration is started immediately after delivery, there is generally entire freedom from breast pain and engorgemnt, as well as distinct depression of lactation. In fact, if suckling is not permitted, lactation, in the clinical sense, does not occur. If the estrogen is employed during the late puerperium, when lactation had already been established, relief from engorgement and subsidence of lactation are

definitely, although not markedly, bettered.

The dosage of estrogen required for the prevention of puerperal breast engorgement is relatively small. There is no need for its intramuscular administration for this purpose. As little as 1 mg. of stilbestrol, or its equivalent in natural estrogen (1.25 mg. of estrone sulfate), given orally three times daily is adequate. Such treatment should be discontinued within ten days and, as exemplified in our clinical experience, it is best withdrawn *gradually* by having the patient reduce the amount taken by one dose daily for several days.

The danger of administering estrogen during the puerperium for too long a period lies in the fact that it is capable of creating hyperplasia of the regenerating endometrium with resultant abnormal uterine bleeding. Such unwanted effects may be avoided by observing the dual precautions of employing a small dose for a limited period of time and discontinuing its use gradually.

4. *Alleviation of the Menopausal Syndrome*: The vast majority of women undergoing the climacteric do *not* require treatment for the menopausal syndrome. When the flushes, nocturnal sweats, and nervous instability are mild, they may be completely mitigated by consultation alone. An explanation of the temporary nature of the symptoms and of their tendency to disappear spontaneously after a variable time, coupled with the prescription of mild sedatives, may be all that is required. However, when the symptoms are marked, there is no more specific therapy than estrogen.

The synthetic estrogens and the orally administered natural estrogens are ideal for menopausal therapy. It is logical to administer the smallest amount necessary to assuage the vasomotor symptoms. Whether one employs a daily dose of 0.5 mg. of stilbestrol, 0.625 mg. of estrone sulfate, or 0.02 mg. of ethinyl estradiol is immaterial. The important rules to be followed are to prescribe the estrogen for only a limited period of time and to withdraw it in a gradual manner. The most prevalent abuse in the usage of estrogen is the prescription of a routine daily dose to be taken more or less indefinitely. This usually results, even in women in whom menstruation had long since ceased, in the occurrence of uterine bleeding. Such bleeding also frequently results following the *sudden* withdrawal of the estrogen. It must be emphasized that the logical objective of estrogen therapy of the menopausal syndrome rests on the fact that it aids in the adjustment of the body to the diminution and final cessation of ovarian function. This aim is entirely lost if the estrogen is not prescribed for measured periods, with repeated attempts to withdraw the hormone entirely. When the withdrawal is gradual, the drop below the critical level does not so readily occur and withdrawal bleeding is avoided.

While postmenopausal uterine bleeding evoked by estrogen is most often caused by faulty dosage and incorrect method of withdrawal, it also occurs in a few of the properly controlled patients. In such cases, the endometrium is peculiarly sensitive to estrogen and it can not be employed without evoking bleeding.

The propensity of estrogen to induce uterine bleeding in the postmenopausal woman is unfortunate. It is presently responsible for a large segment of the neglected instances of carcinoma of the uterus. Prior to the almost universal use of estrogen for the relief of the menopausal syndrome, postmenopausal bleeding was most frequently the result of uterine malignancy. With the current prevalence of estrogen usage, the physician, faced with postmenopausal bleeding, may unfortunately lapse into a false state of security, tending —without proper examination of the patient—to attribute the bleeding to previously administered estrogen. The natural and proper suspicion in all cases of postmenopausal uterine bleeding should be carcinoma of the uterus, and the attending physician should be impelled to inspect the cervix (executing a biopsy if indicated) and to perform diagnostic curettage. That these admonitions are not universally borne in mind is well illustrated by the recorded experience of many cancer clinics. As an example, the authors are privileged to quote some of the recent statistics of the Philadelphia Committee for the Study of Pelvic Cancer, a commission devoted to the study of the histories of patients with pelvic cancer in order to analyze better the factors which delay diagnosis:

From December 1945 until December 1946, the Philadelphia Committee for the Study of Pelvic Cancer uncovered 116 instances in which there had been unwarranted delay in the diagnosis of pelvic cancer. Of these 116 instances, 78 were classified as "physician delay" and the remaining 38 as "physician and patient delay." More significant, perhaps, is the fact that 31 of the 116 patients were given medication, oral or hypodermic or both, in the presence of symptoms suggestive of pelvic cancer, *without examination.* Such a practice, particularly if the patient presents abnormal uterine bleeding, is reprehensible and criminally negligent. It is often a result of the widespread and indiscriminate usage of estrogen prescribed for the relief of menopausal symptoms, as illustrated by the following two case histories selected at random from the 116 mentioned above:

CASE NO. 1

G. N., a white female, aged 41, began to experience irregularities in the menstrual cycle and vasomotor symptoms of the climacteric in January 1946. During February, she bled intermittently for two weeks. When seen by a physician in March, she was advised—without benefit of pelvic examination—that she was "changing" and that she required a few "needles." She received the latter twice weekly for two months with relief of the menopausal symptoms but not of the metrorrhagia. At that time, May 1946, the patient visited another doctor and was told that a small growth was present at "the mouth of the womb," which was removed in the doctor's office. The second physician continued the "injections," in this instance weekly, until the end of June when the patient experienced a sudden vaginal hemorrhage and was hospitalized. The diagnosis was squamous cell carcinoma of the cervix, Stage III.

CASE NO. 2

A. W., a colored female, aged 50, experienced a sudden episode of menorrhagia in the course of her normal menstrual cycle. A physician was summoned, who did not examine the patient but advised rest in bed and the taking of tablets "for the change of life." Menorrhagia was repeated the next month and was followed by intermittent metrorrhagia. After taking the medication for two months and obtaining no relief, the patient saw a second physician who examined her and recommended hospitalization. The diagnosis was squamous cell carcinoma of the cervix, Stage III.

Such histories as these are highlighted examples of the glaring omission of pelvic examination and of the too facile prescription of estrogen. It would seem unnecessary to comment upon the emphatic necessity of pelvic examination prior to the prescription of estrogen. Frequently, mere pelvic examination is not sufficient. Some postmenopausal women, given estrogen after not having menstruated for several years, begin to bleed again. Since it is impossible in such cases to tell whether the bleeding is due to the administered estrogen or to cancer, the physician must adopt one of two courses. He may either wait, observing the effect of the complete withdrawal of estrogen, or preferably, perform an immediate diagnostic curettement. If he elects the former, it is incumbent upon him to be certain that the patient is followed in order to lose as little time as possible should the bleeding not cease promptly. Once a menopausal woman has manifested uterine bleeding following the use of estrogen, it is best that such therapy be avoided entirely. If the persistence of severe menopausal symptoms demands treatment, one may resort to the use of androgen in a dosage calculated to be less than 300 mg. monthly—a dose which ordinarily does not induce masculinization.

Prophylactic use of estrogen, advocated by some for the prevention of symptoms which *may* follow surgical castration, is condemned. Such usage of estrogen appears to be highly illogical and unnecessary. As stated previously, many women who suffer a loss of ovarian function do not develop symptoms and require no therapy. In the others who do, estrogen therapy should be based on the severity of the symptoms.

ESTROGEN ABUSAGE

In addition to certain thoughtless abuses in the *manner* of *administering* estrogen, certain contraindications to its use are often overlooked. The authors avoid the use of estrogen therapy under the following circumstances:

1. *During the Premenopause*: The target for menopausal therapy should be the vasomotor, nervous, and arthritic symptoms in a woman who is *no longer* menstruating. Estrogens are ill-advised in the woman approaching the menopause who has vasomotor syptoms but who also exhibits irregular menstruation. If given at this time, estrogens may easily evoke more irregular bleeding, and may possibly prolong the adjustment of the body to the changing endocrine status. Particularly to be avoided in such women is the treatment of the indefinite "middle-age complaints," manifestations which are only rarely of menopausal origin.

2. *Residual Endometriosis*: In women who develop castration symptoms following the removal of both ovaries because of endometriosis, estrogen is contraindicated. It may be assumed that any patient who had endometriosis which was so advanced and disseminated as to require radical treatment (bilateral oophorectomy) still harbors foci of ectopic endometrium in the remaining pelvic tissues. It is logical to expect that the administration of estrogen to such a woman would stimulate endometrial lesions wherever they may be. If, for example, the residual endometrium lies in the intestinal wall, such functional reactivation might result in serious hemorrhage. It is best, we believe, to avoid the use of estrogen in any woman giving a history of endometriosis. If the castration syndrome cannot be controlled with sympathetic reassurance and mild sedatives, androgen therapy may be employed.

3. *Carcinoma Background*: The tendency to ascribe to estogen the ability of evoking carcinoma in the breast and uterus is based on animal experimentation in which either cancer-susceptible strains or huge dosages of estrogen were employed. It is impossible to administer doses of estrogen in a human comparable to those given in the animal experiments. However, there does appear to be such a thing as "cancer families." While it is generally agreed that estrogen *per se* is not carcinogenic, it cannot be denied that it is a stimulant to growth of tissues which are frequent sites of carcinoma in the female. Moreover, there is no doubt that estrogen will accelerate the rate of growth of an existing carcinoma of either the uterus or breast, as well as any metastasis derived therefrom. If there is a clear-cut history of *multiple* instances of either mammary or uterine cancer in a patient's family background, it would appear safer not to administer estrogen to that patient. It goes without saying that if the patient herself is a salvaged survival from either type of malignant neoplasm she should not be given estrogen.

4. *Palpable Benign Tumors*: Estrogen is the chief stimulant to the growth of the mammary ducts, and because of this physiologic fact, estrogen should not—for any reason— be administered to a patient who exhibits, or who has recently had excised, a fibro-adenoma of the breast.

For similar reasons, the presence of uterine fibromyomas serves as a contraindication to the use of estrogen. Although the quantity of estrogen required to induce growth of an existing fibroid is not known, it is entirely conceivable that even minimal quantities *could* exert an adverse effect.

SUMMARY

1. The therapeutic principles to be observed in the clinical application of the ovarian hormones, estrogen and progestin, have been reviewed.

2. The currently accepted usages of estrogen in the treatment of atrophic vaginitis, amenorrhea, puerperal breast engorgement, and the menopausal syndrome have been discussed. Except in certain instances of amenorrhea, there seems to be no need for the parenteral administration of estrogen.

3. It has been emphasized that estrogen should never be administered without a specific limitation of time. This concept is proved to be especially true in the menopausal patient in whom it is apt to evoke disturbing uterine bleeding, for which curettage is diagnostically imperative. Such bleeding is shown to be less frequent when estrogen is withdrawn gradually.

4. The importance of a careful gyneco-

logic examination, prior to the prescription of estrogen, has been illustrated by the citing of two neglected instances of uterine carcinoma.

5. The contraindications to estrogen therapy have been detailed, including its regrettable use as a "female tonic." The reasons for not employing estrogen in women giving a history of endometriosis, or having a family background of multiple instances of carcinoma of the breast or uterus, or presenting tumors of either the breast or uterus have been reaffirmed.

THE CRUSH SYNDROME IN OBSTETRICS

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In a discussion of the crush syndrome, much attention was focused on this during the bombing of London during the war. At this time there were numerous cases with massive tissue destruction that developed an anuria or oliguria and died from renal failure a few days later. The kidneys of this group of people presented a more or less constant pathologic picture which compared favorably with the previously reported crush syndrome kidneys.

In its relation to obstetrics it has been shown that massive damage affecting the placenta, uterine muscle, or other pelvic tissues may be followed by renal insufficiency and azotemia. Concerning its etiology it may be said that it occurs frequently with abruptio placenta and those abruptios with concealed hemorrhage. Previous pregnancies seem to be a slight predisposing factor, as well as is pre-eclampsia. It has been reported in cases of abortions, and among this group markedly more frequent in the septic abortions. Other traumatic procedures in obstetrics, as extensive laceration of cervix and vagina with hematoma formation, rupture of uterus with massive infiltration of blood into broad ligament and pelvic tissues have been reported to be associated with such syndrome cases. One case of a twisted ovarian cyst with hemorrhagic infiltration into the cyst had had operation delayed for a few days, and presented a similar clinical picture. It is interesting to note that all cases that have been associated with pregnancy have occurred at a time after the placenta has developed.

The clinical sequence of this condition is as follows: First, there is initial tissue damage. Second, in severe cases there is "shock", which may be immediately fatal. In milder cases there may be an absence of shock. Third, in patients who survive shock, there is anuria or oliguria with a urine containing hyaline and granular casts, leukocytes and red blood cells. Fourth, there is a rising blood urea nitrogen which reaches its maximum usually between the fifth and the ninth day and which is followed by death or by an increasing diuresis and eventual recovery which may apparently be complete. The patient succumbs about the seventh or the tenth day, or gets well. A great number of the cases are associated with anemia, probably of a toxic origin (uremia?) and have a rise in blood pressure during the latter part of the course.

PATHOLOGIC PICTURE

In fatal cases the kidneys show three striking pathologic changes. In the first place, the kidneys are enlarged. The cut surface is wet, edematous looking, and drips clear fluid. Secondly, the distal part of the second convoluted tubule and collecting tubules contain dark-colored casts, the pigment of which has been identified as a haem derivative. Thirdly, there are profund histologic changes in the second convoluted tubule and the ascending loop of Henle. Much of the epithelium lining this part of a considerable number of nephrons shows clear evidence of necrosis. This change is particularly intense in microscopic foci usually situated in the boundary zone. In these foci the tubular wall is weakened to such a degree that it must be abnormally permeable to the outward passage of fluid, and at these points there is evidence of tubular collapse with early fibroblastic and histiocytic proliferation in the interstitial tissue. The glomeruli are not involved *per se*.

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TREATMENT

1. Since these patients either die or begin to put out urine within ten days and get well, the treatment is usually conservative.

2. Ample fluid intake in the form of saline and hypertonic glucose to stimulate kidney function.
    a. Alkaline fluids by mouth, prophylactically for recipients of blood transfusions.
    b. Sodium molar lactate to combat acidosis and to prevent precipitation of hemoglobin in tubules by acid urine.
    c. Restoration of blood volume and plasma proteins as is deemed necessary by red blood cell counts and serum protein studies.
    d. Adrenal cortical extract and hypertonic saline has been administered in these cases to cause sodium retention and potassium diuresis, to protect the patients from the convulsive action of the increase of potassium salts in the blood.

3. High doses of ascorbic acid have been given as a diuretic, and vitamin B_1 to prevent or to improve severe vomiting.

4. Decapsulation of kidneys in renal necrosis is a fallacy as there is little evidence in the literature to indicate that this operation is of great benefit. This procedure usually tips the scales toward a more rapid fatal course.

5. The use of a bilateral splanchnic nerve block has proved satisfactory in some cases. The mechanism of this is not exactly understood but is thought to have something to do with the release of the vascular spasm that occurs particularly in renal necrosis. The block should be used early and repeated.

CASE REPORT

C. F. was a 27 year old white female, gravida IV, para II, with last menstrual period February 20, 1946 and expected date of confinement November 27, 1946. During her first pregnancy, the patient had an attack of pyelitis and a marginal placenta praevia which bled at seven months of pregnancy in 1940. A viable baby was obtained, but died thirteen days later (not jaundiced). Patient bled into shock during this delivery, and one 500 c.c. transfusion was given without reaction. Postpartal course was mildly septic (nine-day hospital stay). Second and third pregnancies were uneventful with the delivery of a 10 pound male in 1941 and an 8 pound female in 1943. Neither of these babies showed any jaundice and both have been healthy from birth.

Prenatal course with present pregnancy was complicated by numerous complaints of discomfort confined to lower abdomen, and she had bled about two pads of bright red blood one month before present admission. She had not felt the fetus move during pregnancy. In August, at the fifth month of pregnancy, the patient weighed 143 pounds. On next visit, one month later, weight was 138 pounds. No fetal heart tones were heard. A diagnosis of missed abortion had been made at this time. Blood pressure ranged from 102/70 to 130/80 during prenatal course.

Patient was admitted September 29, 1946 because of brownish vaginal discharge and lower abdominal cramps. After sterile vaginal was done, during which it was impossible to get through cervical os and no cause for discharge was found, she was sent home. Urinalysis at this time was negative. Fetal heart tones were not heard. Blood pressure was 120/82.

October 8, 1946: At 10 a. m., the last admission, patient came into the hospital because she had been passing grossly bloody urine for previous two days. A catheterized urine specimen taken in Admitting Room revealed gross hematuria. Patient was admitted to Urological Service. About 4:30 p. m. patient began to have labor pains associated with slight vaginal bleeding of bright red blood. Bleeding and pains became more severe, and she was transferred to obstetric ward where she was put up for delivery after a type matching and infusion were started. Placental tissue and membranes were protruding through cervical os, and a five-month macerated fetus was delivered; placenta appeared normal.

Impression was a premature separation of a low implanted placenta, five months missed abortion. Total loss of blood was 900 to 1000 c. c. Blood pressure at completion of delivery was 92/60. Five hundred c.c. of plasma was given followed by 150 c.c. of blood which caused a mild chill, and the blood was discontinued. After she had received the plasma, blood pressure was 148/90, pulse 70, respiration 18. Patient continued to ooze blood from vagina over the period of the next three hours, and the blood pressure gradually dropped to 90/50. She was retyped and matched. Five hundred c.c. of plasma was given, and after an inspection of the lower uterine segment and the cervix, the uterus was packed; 500 c.c. of blood was given now without a reaction, and as blood pressure was still around 100-108/60-70, another 500 c.c. of blood was given very slowly. At this time a slight icteric tinge to sclera was noted. Urine during the night was grossly bloody and the output was approximately 30 c.c.

October 9, 1946: One day after admission patient had hemorrhagic areas around gums. Hemoglobin now was 9 grams (63 per cent), red blood cells 4.2 million, white blood cells 25,950, differential count: Polymorphonuclear leukocytes 87 per cent, basophiles 4 per cent, monocytes 5 per cent, lymphocytes 4 per cent, prothrombin 40 per cent of normal; coagulating time 4 minutes 10 seconds, bleeding time 1½ minutes, temperature 101°, pulse 100. Rh typing was done and patient was found to be Rh negative. Husband was called in and was found to be Rh positive. On urologic consultation cystoscopy was done. No urine was found in either renal pelvis. Five c.c. of indigo carmine did not appear from either kidney in 22 minutes. Urinary output was to be stimulated with hypertonic glucose; sodium molar lactate was given; diathermy over kidneys and warm uretal irrigations were done. On medical consultation, nothing significant was added, and intravenous fluids with alkalinization were advised. Penicillin was also given along with large doses of vitamin C intravenously. Since admission, including blood and plasma given, a total of 6400 c.c. of fluids were administered, and an output of 250 c.c., grossly bloody, was evidenced.

October 10, 1946: On second day of admission patient was drowsy and semi-stuporous and was awakened only because she wanted to scratch herself. Blood work now was CO_2 50 volumes per cent, urea nitrogen 53.4, creatinine 4.66, serum proteins 3.2, icterus index 12.5. Urine was grossly bloody with an occasional red blood cell cast. Hematologic survey was hemoglobin 7.2 grams, red blood cells 2.7 million, white blood cells 22,450, platelets 200,000 (direct), hematocrit 24 volumes per cent, hemoglobin 86 per cent; differential count: polymorphonuclear leukocytes 90 per cent, eosinophiles 1 per cent, lymphocytes 9 per cent, reticulocytes 6 per cent, blood type O; clotting time 5 minutes, sedimentation rate 6 minutes per hour corrected, bleeding time 6 minutes, prothrombin time normal.

Comment: peripheral blood picture shows a moderately severe anemia, normochromic and normocytic, marked leukocytosis with shift to left. Bleeding time is just over upper limits of normal, and red cells show a decreased fragility. The significance of this is not readily apparent.

Patient received 500 c.c. of Rh negative blood slowly without reaction. Urinary output averaged 5 c.c. per hour. Total output today was 120 c.c., intake was 4839 c.c. consisting of hypertonic glucose, saline, and molar lactate. Blood pressure ranged from 120-130/60-80. Uterine pack was removed, and there was no further bleeding.

October 11, 1946: On third day patient was semistuporous and had vomited several times; she continued scratching, and had begun to get moderately edematous. Urea nitrogen was 34.8, CO_2 33 volumes per cent, serum proteins 3.2. Urine was still grossly bloody. Thirty five hundred c.c. of intravenous fluids were given plus 525 c.c. of plasma when a chill developed, and the plasma was discontinued. Total output for 24 hours was 268 c.c. Rh studies were done by Dr. Davenport from Baptist Hospital. Blood was Rh negative, Type O, with no evidence of irregular antibodies present. No serologic evidence of Rh involvement was present, but only Rh negative blood as transfusion and sodium molar lactate were advised. Fundoscopic examination was negative.

October 12, 1946: On fourth day patient appeared slightly improved. Urinary output picked up to 10-15 c.c. per hour and late in day to 30 c.c. per hour. Total output for 24 hours was 455 c.c.; intake was 1525 c.c. orally and 2500 c.c. intravenous fluids.

October 13, 1946: On fifth day patient was drowsy but oriented. Edema increased involving face, arms, legs, and trunk. Lungs showed musical rales and few wet rales, after plasma was given. On medical consultation it was thought that an asthmatic allergic reaction to the plasma had occurred. Condition improved after aminophylline was given intravenously. Oral and intravenous intake was 5280 c.c.; output was 272 c.c. Urine was less bloody than previously.

October 14, 1946: On sixth day patient continued to be semi-comatose. Blood chemistry was CO_2 48 volumes per cent, urea nitrogen 53.4, creatinine 8.3, red blood cells 3 million. Urine was less bloody and there were no casts. Intake was 3470 c.c.; output was 415 c.c. Intravenous fluids contained parenamine.

October 15, 1946: On seventh day patient started on high protein tube feedings. Urea nitrogen was 66.8, creatinine 9.7, serum proteins 3.2. Patient received 220 c.c. of fresh Rh negative blood, but she had a chill following this, and temperature rose to 102°. Intake was 3770 c.c. (2500 c.c. intravenously); output was 775 c.c. There were moist rales in both lung bases. Temperature was 100°, and a marked generalized edema was still present.

October 16, 1946: On eighth day there was a marked improvement in the urinary output. Intake was 2410 c.c.; output was 3310 c.c. Urine showed red blood cells 50-60 per high power field, 2-3 white blood cells per high power field, and no casts were present. General condition was the same.

October 17, 1946: On the ninth day urinary output was 5300 c.c.; intake was 2190 c.c. Patient began to improve generally. Urea nitrogen was 49.5, creatinine 9.5, serum proteins 4.45, hemoglobin 9 grams. Patient continued to improve after this, and urea nitrogen and creatinine gradually came down to 23 urea nitrogen on October 24, 1946. Patient made an uneventful recovery after this, and on October 31, 1946 had a Madelener sterilization done. Blood pressure stabilized around 150/90. Rh studies on two living children showed both to be Rh positive.

November 6, 1946: Intravenous pyelography did not show any abnormalties.

December 5, 1946: Postpartal visit showed blood pressure 116/73. Patient is asymptomatic. Urea nitrogen is 15.1 and urine specimen is negative. Patient is now in process of being further investigated by Urology Department.

DISCUSSION

The patient whose case report is presented was a multipara who had a five months missed abortion diagnosed one month before admission, and a normal prenatal course. History of onset was gross hematuria two days before admission and labor, and definitely gross hematuria by catheterized specimen in Admitting Room six hours before labor began. The onset of oliguria at this time was not definitely known, but it was felt that there was a diminished urinary output indicated from the patient's history. This would indicate primary kidney damage, but in the light of subsequent normal intravenous pyelograms, normal two-month postpartal urea nitrogen and urine, the likelihood of primary kidney damage is small and the possibility of a toxic onset of the syndrome at this time is possible. The fact that the patient did have a low lying placenta praevia with partial separation would lend credit to the presence of sufficient amount of tissue damage with resultant renal failure. Patient experienced slight restlessness and a chill during a blood transfusion after 150 c.c. had been given slowly over a period of one hour. However patient had no costovertebral angle tenderness, pain, nor dyspnea. This was not a typical transfusion reaction. Patient also reacted to subsequent units of plasma, which reactions were attributed to her allergic nature and associated with musical rales and wheezes in chest. In hemolytic reactions we do not get an early gross hematuria as in this case. If a hematuria occurs, which is infrequent, it is usually microscopic. The fact that she was icteric early after the blood, and an icterus of 12.6, indicates hemolysis, but again this could be explained on a toxic basis. The possibility of an Rh incompatibility was considered. The patient is an Rh negative individual with an Rh positive husband and two Rh positive children. She had a transfusion (Rh type unknown with first pregnancy). However on blood examination there were no irregular blood agglutinins present, and in the opinion of the pathologist no Rh incompatibility was at fault.

The subsequent course characterized by the gross hematuria, oliguria, gradual rise of urea nitrogen, persistent hypertension during the course, and anemia with sudden profuse diuresis on eighth day and eventual recovery certainly follows the clinical sequence of crush syndrome.

CONCLUSION

Since no pathologic sections were obtained, this patient can be classified only as a renal failure with recovery following a five months missed abortion, which clinically fits the picture of a crush syndrome. This case is presented to stress the possibility of a crush syndrome occurring as a complication of obstetric tissue damage.

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THE REVERSE-TRENDELENBERG POSITION AND EARLY MOBILIZATION AFTER OPERATION*

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The position of the patient in bed following surgery is important because it may prevent or predispose to postoperatve complications. It is the purpose of this paper to present a postoperative position in bed which may help to prevent complications, especially pulmonary and vascular, may serve to promote a favorable mental state, and maintain morale. The position herein discussed may also be of great value for the bed-fast patient, preceding ambulation, and for the geriatric. This is especially true in the aged, who usually remain in one position in bed until told to turn or until they are turned manually.

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*Material for this paper gathered from the wards of Charity Hospital of Louisiana at New Orleans.

In general, there is an unanimity of opinion as to the merits of the Trendelenberg position immediately after operation. There has been considerable variance of medical opinion, however, as to the best position after the patient has reacted from the immediate effects of an operation. The term 'postoperative', as used here, refers to the patient's condition after he has reacted from the effects of surgery and anesthesia, and after the cough reflex has returned.

The three positions favored are: Fowler, semi-Fowler and horizontal in bed. All of these attitudes tend to "freeze" the patient in one position. To overcome this disadvantage, a position called "reverse-Trendelenberg" is recommended.

Trial and error have shown that the best angulation may be obtained by elevating the entire head of the bed eighteen inches. The bed is not broken at the knees in order to prevent kinking of the popliteal and femoral vessels, since kinking may lead to stasis and pooling of blood in the thigh which, in turn, may be a causative factor in thrombus formation. A padded board, fastened at right angles to the mattress at the foot of the bed, gives the patient something solid and firm to push against and prevents the feet from sliding through the bars.

The degree of incline, of course, should suit the individual case. For example, for the aged and weak, only a slight incline may be advisable so as not to tire the patient. Occasionally, if the patient complains that he cannot sleep in the elevated position, the angle of inclination will have to be decreased during sleeping hours. Most patients, however, have no difficulty sleeping in the elevated position. In fact, most cardiopathies have found that they sleep better in the reverse-Trendelenberg position. Cases of shock, hemorrhage, or hypotension should be regarded as specific, though infrequent exceptions to the recommended use of the reverse-Trendelenberg position, and should be treated as such. The position should not be prescribed routinely or dogmatically, since the undue muscular exertion resulting from excessive elevation may, at times, be harmful rather than beneficial. For every patient there is an incline conducive to optimal convalescence and minimal complications, and, judiciously used, the reverse-Trendelenberg position offers several advantages.

The benefits of the position presented are derived from the fact that the patient must exert himself and move about. At various times during the day the patient will slide down gradually in bed against the foot board. In attempting to push himself back, he invariably takes deep breaths and uses all the muscles of the upper and lower extremities, as well as most of the back muscles.

Many factors, surgical and non-surgical, influence the movement of a patient in bed. The surgical factors such as anesthesia, operative technic, parenteral fluids and intestinal suction, are not within the patient's control. The non-surgical factors, psychogenic and physical, are closely interrelated and interdependent, can be partially controlled, and will therefore be considered.

Psychogenic factors deal with the personality of the patient and his reaction to surgical procedure, both before and after operation, and many exert either a favorable or deleterious influence on the patient during convalescence. The surgeon may be able to modify these factors which are usually due to ignorance, fear, or the mistaken apprehension that the wound will be disrupted by motion in bed.

The value of immobilization as a therapeutic measure is deeply ingrained in the lay mind and all too often in the minds of the medical profession. It has been extolled and emphasized for so long that it is almost considered heresy to advise patients to move about in bed. The usual patient, although cautioned and instructed to breathe deeply and move frequently, fails to do so either because of personal inertia or the initial pain caused by the increase in abdominal tension and movement of the abdominal muscles. Even if the patient has good intentions of moving about in bed and breathing deeply, he soon forgets the instructions or finds it difficult to move in the Fowler or semi-Fowler positions. Moreover, in each of these two positions it is almost impossible for the patient to turn on his side and maintain the position with comfort. The reverse-Trendelenberg position was first used to overcome this reluctance on the part of some patients, and forgetfulness on the part of others, to move about frequently and breathe deeply. The results were so satisfactory that it was soon used on most patients.

Those who fear that muscular exertion of the lower extremities upon the foot board might cause a ventral hernia, need only recall that accuracy of suturing in layers and the absence of infection are of primary importance. All other factors are of secondary and minor importance. A sneeze or a cough may produce greater tension upon an abdomnial wound than any movement in bed. The firmness of the suture line is in no way influenced by the degree of movement in bed.

Early mobilization, as herein discussed, offers the advantage of lessened postoperative cardiovascular and pulmonary complications. Normally, the negative pressure within the thorax favors the return of venous blood to the cardiac chambers. Because of the diminished respiratory activity which follows operation, there is an interference with the normal venous return. Postoperatively, the diaphragmatic excursions are lessened, resulting in concomitant diminution in the negative intrapleural pressure, venous stasis, circulatory retardation and diminished respiratory exchange. In addition, in case of a prolonged operation or extensive trauma, cardiac action is diminished and blood velocity and pressure are lessened or lowered. Cardiovascular activity may further be diminished if the patient is permitted to be quiescent during and following an operative procedure. As a result of lying quietly in bed, the muscles of the extremities are relaxed and the return of venous blood to the heart is therefore less rapid than normally. Circulatory activity is further decreased by peripheral vasoconstriction in the capillary bed. Venous stasis occurs and, in turn predisposes to thrombus formation.

The diminished respiratory exchange resulting from the lessened diaphragmatic excursions causes a lowered carbon-dioxide and oxygen exchange. Prevention of this lowered respiratory exchange is important and patients should be made to breathe deeply frequently. By having the patient lie in the reverse-Trendelenberg position, the viscera fall away from the diaphragm. This facilitates better breathing, permits greater excursions of the diaphragm and aeration of the lungs. The tendency to atelectasis is lessened and a greater exchange of carbon-dioxide and oxygen occurs.

SUMMARY

The advantage of the reverse-Trendelenberg position following operation has been presented. The direct benefits are those of early postoperative mobilization and ambulation; indirect benefits are a lowered incidence of pulmonary and cardiovascular complications and an earlier return of the normal functions of the gastrointestinal and the genito-urinary tracts. Early mobility in bed greatly improves the patient's morale, eliminates many unfounded fears and increases his appetite. Morbidity is reduced and convalescence accelerated.

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HOMOLOGOUS SERUM HEPATITIS: CLINICAL IMPLICATIONS*

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It is being recognized with increasing frequency that hepatitis closely resembling infectious hepatitis may follow the injection of various convalescent human serums or vaccines containing human serum, the transfusion of whole blood or plasma, and the use of inadequately sterilized syringes either for venepuncture of parenteral therapy.[1] This form of hepatitis designated as homologous serum jaundice[2] (or homologous serum hepatitis) has been shown to be caused by a virus contained in the blood or blood products administered, or in syringes contaminated with infective blood. Numerous publications pertaining to this disorder have appeared recently and deal with clinical studies or experimental observations in human volunteers. [1, 3] Homologous serum jaundice is much more apt to follow injection of plasma than whole blood,[3e] since plasma, being prepared from large pools, is more apt to contain the icterogenic agent and in addition is given to many recipients.

The various reports dealing with homologous serum jaundice have served to focus attention on its prevention through care in selecting donors, the identification and elimination of blood products shown to be icterogenic, adequate sterilization of syringes, the use of human serum which is not pooled for prophylactic purposes, and the use of only small pools for transfusion purposes.

Homologous serum hepatitis is characterized by an incubation period of 45 to 150 days or more in contrast with infectious hepatitis in which the incubation period is 25 days (maximum 37 days).[3a] (The differences between these two forms of hepatitis are chiefly epidemiological and are listed in table 1). The clinical features of these two diseases are very similar and the histologic changes in the liver identical.[4] There is frequently a preicteric phase of a few days' duration or longer characterized by anorexia, nausea, vomiting, diarrhea, general malaise, weakness, headache, chilly sensations or fever. It is essential that the clinician make special inquiry regarding the presence of this phase, as the patient is so impressed with the appearance of jaundice that he is apt to overlook the symptoms

TABLE 1

DIFFERENCES BETWEEN INFECTIOUS HEPATITIS AND HOMOLOGOUS SERUM JAUNDICE

	INFECTIOUS HEPATITIS	HOMOLOGOUS SERUM HEPATITIS
Incubation period	25 days (maximum 37 days)	45-150 days (or longer)
Oral inoculation (human volunteers)	Effective	Ineffective
Virus detectable in blood	Only during active stages of the disease (not during incubation or convalescent periods)	Demonstrable for weeks during incubation period
Spread by contact	Frequent	Does not occur
Degree of (oral) temperature elevation in pre-icteric phase (human volunteers)	Usually above 100° F.	Usually below 100° F.
Prophylactic value of immune globulin (single dose: 10 c.c.)	Effective if given prior to six days before onset of hepatitis	Does not reduce incidence of hepatitis; retards onset of acute phase

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which preceded the icterus. Abdominal pain is usually improminent, though occasional exceptions may occur. Pruritus is infrequent. The passage of dark urine and light stools precede the appearance of jaundice. Hepatomegaly is generally present with or without associated tenderness. The incidence of splenomegaly is said to be considerably less than noted in infectious hepatitis,[5] in which the frequency is given as 10.5 per cent[6] and 13.5 per cent.[7] Skin eruptions, particularly urticaria and various forms of erythema, are reported to be more common in homologous serum hepatitis than in infectious hepatitis.[8, 9]

The white cell count is usually normal, and anemia does not generally develop as a result of the disease. As in hepatitis in general, the jaundice is mild, moderate, or severe, lasts from three and a half to 12 weeks with an average of four and a half weeks,[5] and biliary obstruction is incomplete. Bile is present in the urine, and there is frequently excess of urobilinogenuria. Among the other laboratory findings, which are also characteristic of infectious hepatitis, are the high incidence of a positive cephalin cholesterol flocculation test and an increased thymol turbidity.

The chief purpose of this report is to emphasize the importance of homologous serum jaundice in the differential diagnosis of jaundice, with particular respect to: (1) metastatic neoplasm of the liver in patients who have had a recent resection of a malignant tumor; (2) stricture of the common duct (or sometimes stone) in patients who have had a recent operation on the biliary tract, usually a cholecystectomy, and (3) neoplasm as a cause of painless jaundice in middle-aged or older people.

Mention has previously been made of two patients, one of whom had pneumonectomy for carcinoma of the lung and the other a resection of a carcinoma of the sigmoid colon.[1] The development of icterus a little over two months and four months respectively following operation suggested the possibility of metastatic neoplasm of the liver with its unfavorable prognosis. The icterus was moderately severe, and a smooth, tender liver was felt to extend two or three finger breadths below the right costal margin. Laboratory tests showed incomplete biliary obstruction with a positive cephalin flocculation test and increased galactose output. In both instances the icterus disappeared and recovery was complete. The following is a brief report of such a case:

CASE No. 1

E. H., a colored male, aged 63, was admitted to the Cincinnati General Hospital on February 14, 1945 and underwent resection of a carcinoma of the transverse colon on March 8, 1945. Between March 8 and March 15, 1945 he received two units of whole blood and three units of plasma, and was discharged from the hospital on April 17, 1945.

He was readmitted to the surgical service on September 29, 1945 with a diagnosis of probable metastatic carcinoma of the liver, because of jaundice which was first noticed on about August 17. There was no history of a preicteric phase, but simultaneous with the onset of jaundice he had noticed indigestion, bloating, epigastric fullness, with vomiting immediately after eating. Abdominal cramps were present, and there was a dull aching pain in the right upper quadrant and periumbilical region. Weakness was prominent, and there was considerable itching of the skin. Physical examination revealed deep jaundice. The liver edge was palpable one finger breadth below the right costal margin; it was moderately firm, smooth, and slightly tender. The spleen was not felt. The white cell count was 4600. The urine contained bilirubin, and Ehrlich's test for urobilinogen was positive in a dilution of 1:16. The stools were light and contained traces of urobilin. The icterus index was 140 and the cephalin flocculation 3+ in 24 hours. There was no direct evidence of recurrence of the tumor, and there was gradual recovery. The patient was discharged from the hospital.

The recognition of homologous serum jaundice in patients who had a recent operation on the biliary tract is particularly important, as failure to do so may lead to an unnecessary secondary operation for supposed common duct stricture or stone. I have seen this problem presented in three patients who in former years would undoubtedly have been subjected to a second operation. The first of these has been described elsewhere[1] but is nevertheless included here:

CASE No. 2

J. R., aged 63, was first admitted to the Cincinnati General Hospital on March 14, 1946, with a complaint of severe epigastric pain radiating to both upper quadrants, with nausea and vomiting. He had had recurrent bouts of epigastric pain since June, 1945. There was slight icterus; the maximum icterus index was 25. The clinical diagnosis was cholecystitis with common duct stone. He was given 1 or 2 units of plasma on March 24, 25, 26, 27, 29, and 30. On March 28 cholecystectomy and choledochostomy were performed, with removal of a common duct stone. The postoperative course was uneventful, and the patient was discharged from the hospital on April 22.

He was readmitted on June 7. Ten days before he had had a shaking chill followed by headache, anorexia, epigastric discomfort, nausea, and fever. Five days later, the fever disappeared and the urine became dark. At that time his wife noted that he was jaundiced. Physical examination revealed marked icterus. The liver extended four finger breadths below the right costal margin; the surface was smooth and not tender. The spleen was not palpable. There were no vascular spiders. The white blood cell count was 8100, with 76 per cent polymorphonuclears, 19 per cent lymphocytes, and 5 per cent monocytes. The urine contained bilirubin and an excess of urobilinogen. The icterus index was 98; cephalin flocculation 4+ in 24 hours; thymol turbidity 20 units; alkaline phosphatase 7 Bodansky units. The stools at first were light yellow and then became brownish and contained urobilin.

Diagnoses first considered were stricture of the common duct and residual stone in the common duct. However, in view of the administration of plasma a little over two months before the appearance of icterus, the presence of a rather characteristic preicteric phase, and the results of the laboratory tests, a diagnosis of homologous serum jaundice was made. The patient's condition steadily improved. On June 28, the thymol turbidity was 4 units and the cephalin flocculation 1+ in 48 hours. He was discharged from the hospital on July 11, 1946. Another case presenting this problem follows:

CASE No. 3

A. B., a white female, aged 31, was admitted to the Cincinnati General Hospital on September 9, 1946 with a diagnosis of acute intestinal obstruction. In June, 1946, a cholecystectomy was performed at another hospital; the gallbladder was small and contracted and contained numerous stones. On September 10, 1946, laparotomy was performed and an adhesion about the terminal ileum was released. She was given 1 unit of plasma on September 10, 1946 and 1 unit of whole blood on September 11. She signed her release from the hospital on September 17, 1946.

She was readmitted to the surgical service of the Cincinnati General Hospital on January 29, 1947, with a diagnosis of stricture of the common bile duct. Six weeks prior to admission she began to experience headache, malaise, anorexia, and weakness. Chilly sensations had been present, and she thought that she had fever. Three weeks before admission she became jaundiced, and she noticed that her urine was dark and her stools light. At the same time, she experienced a constant, boring pain in the lower abdomen and right upper quadrant and vomited once. There was generalized itching. She stated that she was two months pregnant.

Physical examination revealed the presence of icterus. The liver extended four finger breadths below the right costal margin; the surface was smooth and not tender. The spleen was palpable two finger breadths below the left costal margin. There were no vascular spiders. The white blood cell count was 7900 with 78 per cent polymorphonuclears and 22 per cent lymphocytes. The urine contained bilirubin and an excess of urobilinogen. The icterus index was 80; cephalin flocculation 3+ in 24 hours and 4+ in 48 hours; thymol turbidity 15 units.

In view of the administration of plasma and blood three and a half months before the onset of jaundice, the occurrence of a rather characteristic preicteric phase, and the results of the laboratory tests, a diagnosis of homologous serum jaundice was made, and it was advised that surgery be deferred. The patient then signed her release from the hospital. She was seen again on February 21, 1947. The jaundice had almost disappeared; the icterus index was 15. The liver had receded to two finger breadths below the right costal margin; the spleen was still palpable. The cephalin flocculation was negative in 24 hours, and the thymol turbidity 4 units.

Another case is of interest in that it probably represents a combination of common duct stone and serum hepatitis, which is also important to recognize in order properly to defer operation until the acute hepatitis has subsided:

CASE No. 4

E. G., a 59 year old white male, was admitted to the surgical service of the Cincinnati General Hospital on April 6, 1946, with the diagnosis of chronic cholecystitis with lithiasis and possible common duct stone. Because of the presence of pulmonary tuberculosis, cholecystostomy was done with drainage of the common duct. Beginning April 11, 1946, he was given 1 to 2 units of plasma daily. On July 29, 1946, he noticed the appearance of jaundice four days after he began to pass dark, brown urine. With the appearance of icterus there was anorexia and a sudden severe cramping pain in the center of the back and right upper quadrant, lasting 10 hours, during which he vomited once.

Physical examination revealed the presence of jaundice and an external biliary fistula. The liver extended two finger breadths below the right costal margin, and its surface was tender. The spleen was not palpable. There were no vascular spiders. The white blood cell count was 8200. The urine contained bilirubin and urobilinogen, and urobilin was present in the stools. The icterus index was 100; the cephalin flocculation 3+ in 24 hours; the thymol turbidity 12 units. The icterus gradually decreased, and the patient was discharged on August 30, 1946. When last heard from on February 22, 1947, there had been no return of the icterus or of the abdominal pain.

It is quite possible that this patient had a common duct stone (or stricture) in addition to homologous serum jaundice, in view of the presence of jaundice at the time of his first operation and the subsequent development of a biliary fistula, and in view of the sudden, severe abdominal pain which lasted 10 hours and radiated to his back. In favor of homologous serum jaundice are the appearance of his second bout of icterus three and a half months after the plasma transfusions, the presence of dark urine for four days before the sudden, severe abdominal pain, and the results of the laboratory tests.

Flickinger and Masson[10] have recently pointed out that pain is absent in about two-thirds of patients with common duct stricture and that chills and fever occur in about 44 per cent. These clinical features may increase the difficulty in differentiating homologous serum jaundice from common duct stricture, although the fever is apt to be higher in the latter disorder. Neefe, Stokes, and Gellis[11] noted that during the preicteric phase the temperature was usually below 100° F. in their experimental subjects with homologous serum hepatitis. On the other hand, the occurrence of jaundice within a few days after operation, as reported in 41 per cent of Flickinger and Masson's patients with common duct strictures, would naturally exclude homologous serum jaundice. Similarly, the occurrence of a biliary fistula following operation which was noted in 58 per cent of their cases would argue strongly for the diagnosis of stricture.

It is well also to consider homologous serum jaundice in older patients who were previously admitted to the hospital for one cause or another, such as fractures for example, and who were given either plasma or blood at the time of their original admission. Because of their age, neoplastic jaundice is apt to be suspected. While it is recognized that hepatitis may occur at any age, the history of previous transfusion with blood or plasma may prove very comforting to the physician in his diagnosis of serum jaundice in a middle-aged or older patient.

CASE No. 5

F. S., a 62 year old white male, was admitted to the orthopedic service of the Cincinnati General Hospital on May 9, 1946. On June 29, 1946, an osteotomy was performed for non-union of an intertrochanteric fracture of the femur. He had received one unit of whole blood on June 20, 1946, 1 unit of plasma and another unit of whole blood on June 29, 1946. Jaundice was first noticed on October 26, 1946, while he was in the hospital. The icterus was not preceded nor accompanied by any other symptoms except for diarrhea which developed later. The skin and sclerae were definitely icteric. When the patient was first seen, the abdomen could not be examined because of the presence of a plaster cast, but two months later the liver edge extended three finger breadths below the right costal margin; it was smooth and not tender. The spleen was not palpable. The urine contained bile pigment and an excess of urobilinogen. There was an excess of urobilinogen in the stools. The icterus index was 48; the cephalin flocculation 2+ in 24 hours and 3+ in 48 hours; the thymol turbidity was 6 units; the alkaline serum phosphatase 3.4 Bodansky units. A transthoracic needle biopsy of the liver on November 20, 1946 was read by the pathologist as "infectious hepatitis—regenerative phase."

CASE No. 6

W. S., a 48 year old white male, was admitted to the surgical service of the Cincinnati General Hospital on August 9, 1946 for fractures of the right sixth, seventh, eighth, ninth, tenth and eleventh ribs and right scapula. On August 9, 1946, he was given 1 unit of whole blood. He was readmitted to the hospital on October 1, 1946. On September 23 he had noticed the appearance of jaundice. For four days preceding jaundice, he experienced anorexia, severe nausea especially after eating fatty foods, and constipation. With the onset of the icterus, he noticed that his urine was dark and his stools light. There was no abdominal pain, fever, chill, pruritus, or vomiting.

Physical examination revealed the presence of mild icterus. The liver edge extended two finger breadths below the right costal margin; the surface was smooth and not tender. The spleen was not palpable. The urine contained an excess of urobilinogen and urobilin was present in the stools. The one minute serum bilirubin was 2.2 mg. per cent, the total bilirubin 4.8 mg. per cent. The icterus index was 32; the cephalin flocculation 3+ in 48 hours; the thymol turbidity 4 units. The white blood cell count was 9200 with 64 per cent polymorphonuclears, 30 per cent lymphocytes and 6 per cent monocytes. The icterus gradually disappeared, and the patient was discharged on October 10, 1946.

A brief outline of the six cases herein reported is given in table 2. Since we have been on the alert for cases of homologous serum jaundice, my associates Drs. William Molle and Carl Kumpe and I have seen 11 cases in a little more than a year. It is our belief that many cases are overlooked because the condition is not usually considered in the differential diagnosis of jaundice, and it is quite probable that needless operations have been performed on this account. It is essential that the physician keep homologous serum jaundice in mind whenever he is asked to see a patient with icterus, regardless of the age of the patient, particularly if there is a history of an illness or an operation two to five months before the development of jaundice, even if the operation was performed on the biliary tract. His suspicions should be confirmed by recourse to the cephalin cholesterol flocculation and thymol turbidity tests.

Since this paper was written, C. J. Watson[12] stated in a visit to the Cincinnati General Hospital that he has seen homologous serum jaundice develop in three patients following operations on the biliary tract for chronic cholecystitis and cholelithiasis with common duct stone. These patients were jaundiced at the time of their operation, and the common duct was opened in each instance. The clinician was naturally misled into a diagnosis of common duct stone or stricture when jaundice recurred following operation. Watson accordingly emphasized the importance of distinguishing homologous serum jaundice from common duct stone or stricture.

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TABLE 2
SUMMARY OF CASES

Case #	Age	Sex	Col.	Primary Diag.	Type & Amt. Transfusion	Interval bet. Trans. & Onset of Jaundice	Preicteric Phase	Symptoms Icteric Phase	Hepatomegaly	Splenomegaly	Icterus Index	Ceph. Choles. Floc.	Thymol. Turbid.
1	63	M	C	Ca. of trans. colon	2 units whole blood; 3 units plasma	5½ months	Absent	RUQ pain, jaundice, indigest., weakness, itching	1 f.b. below costal margin, tender	Absent	140	24 hr. 3+	....
2	63	M	W	Cholecystitis with common duct stone	7 units plasma	2½ months	Malaise, chill, fever 102°	Painless jaundice, brown urine, yellow stool	4 f.b. below costal margin, tender	Absent	98	24 hr. 3+	20 units
3	31	F	W	Cholecystitis & cholelithiasis After-operation - intest. obstr.	1 unit whole blood; 1 unit plasma	3½ months	3 wks. malaise, headache, anorexia, weakness	RUQ pain, jaundice, itching	4 f.b. below costal margin, not tender	2 f.b. below costal margin	80	24 hr. 3+	15 units
4	59	M	W	Chronic cholecystitis with lithiasis	7-10 units plasma	3½ months	4 days dark brown urine	RUQ pain, jaundice, anorexia, light stools	2 f.b. below costal margin, tender	Absent	100	24 hr. 3+	12 units
5	62	M	W	Non-union of intertrochanteric fracture of femur	2 units whole blood; ½ unit plasma	4 months	Absent	Painless jaundice, acholic stools	3 f.b. below costal margin, not tender	Absent	48	24 hr. 2+ 48 hr. 3+	6 units
6	49	M	W	Fracture of rt. 6th, 7th, 8th, 9th, 10th, 11th ribs & rt. scapula	1 unit whole blood; 2 units plasma	1½ months	4 days nausea anorexia, constipation	Painless jaundice, dark urine, light stools	2 f.b. below costal margin, not tender	Absent	32	48 hr. 3+	4 units

ECHINOCOCCUS INFECTION (HYDATID DISEASE) IN LOUISIANA

J. C. SWARTZWELDER, Ph. D.
NEW ORLEANS

According to a report by Magath[1] at least 519 cases of hydatid disease were reported prior to 1940 in Canada and the United States since the first case seen in 1808. It was pointed out by Magath[1] that 95 per cent of the cases, in which the nationality was known, have occurred in immigrants. In contrast, a review of 15 cases of hydatid infection which have occurred in Louisiana reveals that 10, or two-thirds, of these have been in native citizens of the United States.

In 1938 Sawitz[2] summarized the cases of Echinococcus infection in Louisiana which had occurred or were reported up to that time. His summary included eight cases, of which five were previously unreported. The present report includes seven additional cases of hydatid disease which have occurred in Louisiana since 1938, three of whom were native-born residents of the state. A tabulation of the known cases of hydatid infection which have been diagnosed in Louisiana to date is presented in the accompanying table. Two additional unreported cases were diagnosed as Echinococcus infection in 1908 and 1916 in New Orleans. However, since the data on the basis of diagnosis are not complete and as I have no personal knowledge of these two cases, they are not included in this report. Since, with few exceptions, the cases reported in this entire series were patients at the Charity Hospital of Louisiana at New Orleans, it is probable that cases of hydatid disease may have been diagnosed in other parts of the state and have not been reported in the literature.

Eleven of the 15 cases of hydatid infection which have occurred in Louisiana have been diagnosed since 1932. It is probable that an increase in the knowledge of the infection among physicians and improvement in the methods and facilities for diagnosis may account to a great extent for the recent increase in the reported cases. Ten of the 15 cases occurred in individuals born in the United States, four of whom were negroes. This emphasizes that human infection with this endemic disease may be acquired in this country. It is possible that some of the infections in immigrants who

TABLE I

CASES OF ECHINOCOCCUS INFECTION WHICH HAVE OCCURRED IN LOUISIANA

Author	Date of diagnosis	Date reported	Race	Age	Sex	Location of cysts	Nativity (U. S. or foreign)
Osler[3] (Ogden)		1882			F	Liver	Foreign
Smith[4]	1919	1928	W	44	F	Liver mesentery	Native
Miller and Collins[5]	1935	1937	C	22	F	Uterus	Native
Sawitz[2]	1906	1938	W	36	F	Liver	Native
"	1913	1938	W	64	F	Liver	Native
"	1932	1938	W	38	M	Liver	Native
"	1937	1938	C	53	F	Breast	Native
"	1937	1938	W	72	F	Liver	Native
Writer	1938	Not previously reported	W	30	M	Liver	Holland (resident of U. S. for 18 years)
"	1939	"	W	59	F	Liver	Italy
"	1939	"	C	54	M	Liver	Native
"	1939	"	W	60	M	Liver	Italy (resident of U. S. for 45 years)
"	1940	"	W	23	M	Brain	Native
"	1941	"	C	34	M	Brain	Native
"	1943	"	W	51	M	Liver	Italy (resident of U. S. for 37 years)

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†From the Department of Public Health of Louisiana State University School of Medicine and the Charity Hospital of Louisiana at New Orleans.

resided in this country for several decades before diagnosis of the disease also may have been acquired in this country.

Hydatid disease of man is caused by the larval stage (hydatid or echinococcus cyst) of the tapeworm, *Echinococcus granulosus*. The minute adult tapeworm parasitizes the intestine of dogs and certain other carnivores. The source of infection for man is usually considered to be through contamination of food or drink with excreta of infected dogs, which contains the infective eggs of the parasite. Cattle, sheep and hogs are the common hosts of the larval stage (hydatid cyst) in nature. The infection reaches a cul-de-sac in man. Dogs or other carnivorous animals derive their intestinal infection with the adult tapeworm through ingestion of the carcasses of cattle or other animals harboring the hydatid cysts. Reports of infection with *E. granulosus* in dogs in this country are rare. No infections with this parasite were reported by Hinman and Baker[6] from a helminthologic survey of 1315 dogs from New Orleans. Apparently no infections in dogs have been reported from Louisiana, despite the occurrence of hydatid cysts in cattle and pigs in this area. Riley[7] has reported natural infection of wolves with the adult tapeworms and the occurrence of hydatid cysts in moose in Minnesota, which suggests that wild mammals also may serve as reservoir hosts of the parasite.

The diagnosis of the seven previously unreported cases in this series was based upon demonstration of the cysts and their contents after surgical removal, by aspiration of the cyst contents, or from postmortem examination. Intradermal tests were also done on two of these cases, one employing hydatid fluid and one using a substitute tapeworm antigen. Both elicited positive cutaneous reactions. Although the demonstration of typical scolices and hooklets provides the most certain evidence of Echinococcus infection in man, there are other valuable and practicable diagnostic tests available to help establish the diagnosis of the disease.[1, 8, 9] These include the intradermal test of Casoni which employs sterile hydatid cyst fluid from animals or from man as antigen, and similarly a cutaneous test[9] using extracts of certain other species of tapeworms commonly found in lower animals, which are perhaps more readily procurable than hydatid fluid. Recently the Biological Division, Lilly Research Laboratories, Indianapolis, Indiana, has announced that Echinococcus antigens extract for skin testing and for complement-fixation for hydatid infection are available for distribution on an experimental basis. The intradermal reaction may remain positive for many years after complete removal of the cyst. Complement-fixation and precipitin tests employing these antigens are also of diagnostic value. The latter serological tests have, in general, a reported accuracy of about 85 per cent, but represent specialized procedures which are not available in small laboratories. Since some danger may attend diagnostic cyst puncture and since diagnosis of hydatid disease is often made initially by exploratory operation, the advantage of use of the intradermal test prior to operative procedure is obvious. Physical findings, such as evidence of a cyst or mass, particularly in the region of the liver, and hydatid thrill, the use of x-ray for visualization of the cyst and performance of a differential leukocyte count for presence of eosinophilia (which occurs in about one-fourth of the cases) may contribute to the diagnosis when such nonspecific evidence can be elicited.

SUMMARY

A total of 15 known cases of Echinococcus infection (hydatid disease) in man, including seven relatively recent cases reported in this article, have occurred in Louisiana. Ten of the infected individuals were born in this country and four of these were negroes. This indicates the native origin of the infections and emphasizes the possibility of acquiring the disease in this area. Whereas Echinococcus infection has been found more often in native-born residents of Louisiana than in immigrants, 95 per cent of the total cases of hydatid disease reported from the United States and Canada prior to 1940, in whom the nationality was

known, have occurred in immigrants. The value of employing intradermal and complement-fixation tests with either sterile hydatid fluid or substitute tapeworm antigens in an effort to provide a diagnosis prior to operative procedure has been pointed out.

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THE PART OF THE GENERAL PRACTITIONER IN THE MANAGEMENT OF VESICAL NECK OBSTRUCTION*

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Most cases of vesical neck obstruction come to the urologist through the hands of the general practitioner. Many of them come because the general practitioner knows exactly what is wrong and what should be done. That's as it should be. Other patients come merely because there are urinary symptoms and the general practitioner does not know what is wrong or what should be done. That's not as it should be—he should know what's wrong and he does not need a cystoscope to find out. Other persons in need of surgical interference remain unduly long in the hands of the general practitioner, taking urinary tract antiseptics and prostatic massages to no good purpose. That too is not as it should be. And finally, some few patients never do get to the urologist: either purposeless palliation is pursued to an unnecessary fatal outcome or surgical management is accorded by incompetent hands. That certainly is not as it should be.

I say these things as one who for 20 years has had a remarkably happy relationship with a host of general practitioner colleagues throughout the Northwest—men who have given me a large and interesting referred practice in my special field. That is not to say that these men in general practice and I have jointly managed our task to the best advantage of our patients. I am only too well aware of my own deficiency in the matter—but my shortcomings and my part in the job are not my subject. Also I have come to see the shortcomings of the general practitioner and to know that his part in our joint enterprise can be improved. That is my subject.

In the management of vesical neck obstruction the proper part of the general practitioner lies in diagnosis, in preoperative management, in directing the patient to a competent surgeon at the proper time and in postoperative care.

For the surgical part of management many of these patients must go considerable distances from their homes to hospitals in larger centers. Much time and expense can be avoided if the patient is kept ambulatory under the care of his family physician while a definite diagnosis is made and during the period of preoperative treatment and for much of the postoperative care. All of these—diagnosis, preoperative treatment and much of the postoperative care—can and should be done by the patient's family physician to the end of avoiding needless loss of time and unnecessary hospital expense.

When a patient presents himself with urinary symptoms, the general practitioner can and should promptly find the answers to the following questions:

1. Is the trouble due to vesical neck obstruction?
2. What type of vesical neck obstruction is present?

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3. Is surgical treatment required at once?

4. May palliative treatment be employed safely with postponement of operation?

5. Is the patient ready for immediate operation or will he be made a better surgical risk by preoperative treatment directed by the general practitioner?

Let's take the questions in order.

IS THE TROUBLE DUE TO VESICAL NECK OBSTRUCTION?

In considering this question it is a great mistake and will lead to blunders to think that benign prostatic hypertrophy and prostatic carcinoma in the elderly male are the only causes of vesical neck obstruction. There are other causes, less frequent it is true, but for the patient who has such obstruction they are more important than benign hypertrophy or carcinoma. And do not lose sight of the fact that vesical neck obstruction does occur in young individuals, that it is often present without palpable enlargement of the prostate and may occur in females.

From the history alone you can usually come close to knowing whether or not there is obstruction. The typical symptoms in order of onset are:

1. Hesitancy in starting the stream. At first this may affect only the first urination in the morning. Later it is noted at the urinations during the night and still later it is always present.
2. Nocturia. This may be the only symptom.
3. Increased daytime frequency.
4. Urgency.
5. Decrease in the size and force of the stream.
6. Interrupted urination.
7. The sensation of incomplete emptying.
8. Complete retention of urine.

The appearance of these symptoms one after another—some or all of them—and final presence of most of them means only one thing: vesical neck obstruction.

The impression as to the presence or absence of vesical neck obstruction gained from the history will be verified at once by a very simple and cursory kind of physical examination and in nine cases out of ten without the use of the cysto-urethroscope.

The patient should be examined at a time when there is a real desire to urinate.

The act of urination in the standing position is observed. The patient is instructed to empty the bladder completely. If there is obstruction at the neck of the bladder there is usually some degree of hesitancy in starting the stream. The size and force of the stream are noted. Even with well developed obstruction the first part of the stream, when the bladder has tone because of its filled condition, may be of good force. An abnormally prolonged terminal decrease in force is significant. After voiding to empty the bladder a catheter is immediately passed. This must be done with the patient in the erect position. The residual urine contained in a very atonic bladder will not evacuate through a catheter with the patient in the recumbent position. For the same reason residual determinations should follow voiding to empty with the patient in the erect position. The presence of residual urine is an important finding. Residual urine in excess of 30 c. c. is positive proof of vesical neck obstruction. However, absence of residual urine by no means rules out obstruction. If the bladder has reacted to obstruction by concentric hypertrophy the powerful trigone may be capable of pulling open the obstructed vesical neck and the well compensated detrusor may force complete evacuation.

When a history containing significant symptoms of obstruction is combined with the presence of residual urine one may say with confidence that there is vesical neck obstruction. This becomes a certainty when the obstructive symptoms and residual urine are combined with physical signs of the obstructing lesion.

The physical signs of the obstructing lesion contain the answer to the second question:

WHAT TYPE OF VESICAL NECK OBSTRUCTION IS PRESENT?

The causes of vesical neck obstruction to be borne in mind are in order of frequency:

1. Benign prostatic hypertrophy.
2. Carcinoma of the prostate.
3. Prostatic bar.
4. Contracture of the vesical neck.
5. Inflammatory swelling of the vesical neck—acute prostatitis in the male, acute urethritis in the female.
6. Disturbances of innervation, the result of organic central nervous system disease, or functional disturbances as in postoperative retention.

This is not a very long list to be kept in mind. If the physiologic-pathologic approach is employed in taking the history and examining the patient very promptly the condition present will fall under one of these designations.

Much is learned from the rectal examination in men, and vaginal touch in women. Obstructions due to acute inflammatory change produce a boggy swelling of the prostate and tenderness to pressure on the gland. In women there is tenderness to pressure against the urethra and bladder neck. With postinflammatory vesical neck contracture in women the urethra when palpated against the pubic arch is much thickened and infiltrated. Postinflammatory vesical neck contraction in men is usually associated with a small, somewhat irregular prostate which lacks the normal resilience and is well described as "gristly." "Prostatism without a prostate" the old urologists called it.

Benign enlargement of the prostate may be almost entirely intravesical and give little or no enlargement palpable by rectal examination. In most cases, however, some degree of enlargement is apparent by rectal examination. The gland is smooth, usually firm, but resilient. The globular lateral lobes form a sulcus on each side with the pubic ramus and between the two lobes is a well defined median furrow covered by the uninvolved posterior lamina or lobe. The attachments are elastic and the gland is very slightly movable.

These characteristics on rectal examination sharply distinguish benign prostatic hypertrophy from carcinoma and make of the rectal examination the most valuable item we have in differentiation of the two conditions.

The typical carcinomatous gland is stony hard. The surface is irregular or even nodular. Distinct lateral lobes are not distinguishable and the median furrow between them is obliterated. The sulci between the gland and the pubic rami are filled out and obvious growth in these directions may be found. Fixation is so firm that no movement can be detected. The differential diagnosis between a carcinomatous gland and benign enlargement in which infection and cicatrization have occurred may be difficult.

From the history and physical examination thus far, and keeping in mind the changes characteristic of the different obstructing lesions, one can usually say in general that there is vesical neck obstruction and say in particular that the obstruction is due to benign prostatic hypertrophy, or to carcinoma of the prostate, or to an acute inflammatory process: that is acute prostatitis or prostatic abscess in the male, or acute urethritis and cystitis of the vesical neck in the female.

When the physical changes characteristic of these conditions are absent, the remaining possibilities are postinflammatory contracture of the vesical neck or disturbed innervation—both of which may occur in either male or female.

The first steps in the further differentiation should be careful re-inquiry into the history and a cursory kind of neurologic examination.

A history of gonorrhea or more important, postgonorrheal prostatitis with persisting "morning drop," or perineal discomfort, are almost regularly obtained in cases of bladder neck contracture in men. In women the similar condition has usually been preceded by oft repeated attacks of "cystitis" which actually have been exacerbations of urethritis.

If the condition present does not fall into any of the mentioned forms of pathologic tissue change affecting the bladder neck, then disturbed innervation is the only remaining explanation.

A history of neurologic disturbances in general should always be looked for. Further inquiry may elicit a history of chancre and the latter manifestion of tabes, such as ataxia and other symptoms of the disease. Or there may be complaint of paresthesias over the saddle area or other neurologic disturbances to indicate other forms of central nervous system disease. Any of these suggest the possibility of disturbed bladder innervation.

The cursory neurologic examination is extremely simple and embraces only pupillary reflexes, station and equilibrium tests, knee kicks, plantar reflex and sensation over the saddle area.

The presence of any significant neurologic signs disclosed by this examination suggest the possibility of disturbed innervation of the bladder and urethral sphincters as cause of the obstructive symptoms and residual urine. A neurologic consultant may give valuable aid in such cases. The possibility of symptoms being due to neurologic disease must be borne in mind constantly—even in cases where anatomic change in the vesical neck seems obviously to be responsible.

Finally in a very small number of cases it may be impossible from the history and ordinary methods of examination to say with confidence what causes the obstruction. In these few cases there must be resort to the cysto-urethroscope.

The next question is:

IS SURGICAL TREATMENT REQUIRED AT ONCE?

The answer to this is simple. It is determined by the grade of obstruction and severity of symptoms. The degree in which retention has impaired renal function and the general physical condition do not necessarily have bearing on the question of immeate operation—except to contraindicate it in many cases.

Obviously the individual with complete retention of urine and in sufficiently good general physical condition for immediate operation sould have the obstruction surgically relieved with as little delay as possible.

Likewise, the individual with severely distressing symptoms and instantly a good surgical risk should be afforded the relief of surgical removal of the obstruction without delay.

A very large residual urine in the presence of sufficiently good physical condition to permit operation at once, or in the immediate future, is in a general way indication for such management. Postponement of operation in such cases usually leads to further impairment of renal function and deterioration of the general physical condition. These convert a good risk into a poor risk. However, if the patient's family and business matters, or other surrounding circumstances demand a little delay this may appear appropriate. Such phases of the problem provide good exercise of the intimate relationship usually existing with the general practitioner or family physician. During the period of delay the patient should be under pretty careful observation. Even it may be permissible or advisable to have him report once or twice a day for catheterization or even let him wear a self-retaining bag catheter through which the bladder is completely emptied several times during the 24 hours. During the period of postponed operation these measures will avoid progressive damage, provided repeated over distentions of the bladder are avoided. They should be accompanied by the administration of one of the sulfonamide drugs to control the inevitable infection of an indwelling catheter or interval catheterization.

Very rarely profuse bleeding from a very much enlarged and greatly engorged benign hypertrophy is an indication for immediate surgical interference. Contrary to popular belief, hematuria is much more suggestive of benign hypertrophy than of carcinoma.

The next question is:

MAY PALLIATIVE TREATMENT BE EMPLOYED SAFELY AND OPERATION POSTPONED?

The patient is entitled to an honest answer to this question. I am sure he always gets it. But even an honest answer may be a prejudiced answer. In respect of this matter I believe the prejudice of a general practitioner is fairer to the patient than the prejudice of the urologist.

The individual with only a small residual and not very troublesome symptoms, even though he has outspoken vesical neck obstruction, may safely postpone operation—sometimes indefinitely.

It may seem pretty certain that eventually operation will be required. In view of that the urologist usually feels that the sensible thing is to change the operation from a prospect to a memory and at the same time give relief of the trifling symptoms. But after all it is the patient's problem. If he prefers to put up with his not too distressing obstructive symptoms, have massages from time to time when the bladder irritation is increased, take sulfonamides off and on for control of infection and cooperate in the other details of management, it is his own affair. The family physician familiar with the patient's nervous make-up and factors in his domestic and business life which incline him to postponement is much better qualified than the stranger urologist to help in this decision. In his palliative and expectant management of the situation the general practitioner should bear in mind that obstructive lesions are usually progressive. Accordingly, frequent determinations of residual urine should be made. If residual increases to substantial proportions or the obstructive symptoms are not well controlled and there are disturbance of sleep and inroads on the general physical condition, palliation should be abandoned in favor of surgical relief.

The next question is:

IS THE PATIENT READY FOR IMMEDIATE OPERATION, OR WILL HE BE MADE A BETTER SURGICAL RISK BY PREOPERATIVE TREATMENT DIRECTLY BY THE GENERAL PRACTITIONER?

The answer to this question lies in careful appraisal of the general physical condition with respect to cardiovascular system and other items apart from the urinary tract, and in appraisal of the degree of renal function impairment which has resulted from the obstruction.

It is eminently appropriate that the general practitioner should make these appraisals, arrive at a decision from them and carry out the needed preoperative treatment if this is indicated.

Many of these patients in need of preoperative treatment whether it be for impaired general physical condition, the result of cardiovascular trouble or other constitutional disease, or because of the inroads of urinary obstruction on renal function with nitrogen retention, do much better at home under the care of their familiar family physician than in a hospital under the care of the stranger specialist and his consultants.

It is not for me in my narrow specialty to suggest to you what should be done for hypertension, cardiac decompensation, other forms of cardiovascular disease, diabetes and other constitutional illnesses. I always depend upon consultants for this management. You know better than I how to deal with these conditions and improve the condition of the patient impaired by them.

The impaired physical condition and poor surgical risk which indicate postponement of operation and require preoperative treatment are often solely the result of renal function impairment. They are indicated by the typical clinical features of uremia and verified by blood chemistry determinations. Urologists have always felt that the management of this condition and its correction is their special province. We do pretty well with it, but there is no reason why the family physician who will devote himself to the problem cannot handle it just as well. In this way long periods of hospitalization and much expense can be avoided.

The clinical features of extensive renal impairment are familiar to all of you and need no comment here.

The first essential in the management of renal impairment resulting from vesical neck obstruction is drainage. In the vast majority of cases this can be provided by either interval catheterization or constant drainage by an indwelling catheter. Only rarely is drainage by suprapubic cystostomy required because of difficult catheterization or intolerance to the indwelling catheter.

With long standing obstruction and impairment of renal function there always is a very marked decrease in the specific gravity of the urine. Determinations of specific gravity may be made from day to day. With the patient on a constant fluid intake, improving renal function will be indicated by a rising specific gravity.

Functional improvement can be measured more accurately by sending in to a laboratory from time to time samples of blood for determination of the degree of nitrogen retention. For this only the blood urea nitrogen need be reported. Changes in this value will indicate accurately changes in the renal function. Other indicators of improved renal function are increasing appetite, gain of weight, increasing alertness and interest in life, improvement of the usually present anemia as indicated by color of mucous membranes and improvement in the patient's condition in general.

If interval catheterization is employed, the catheterizations should be done frequently enough to avoid any discomfort from bladder filling, and at all events so frequently that not more than 400 c.c. of urine are found in the bladder. The frequency of catheterization must be proportioned to the fluid intake—or more exactly to the urine output.

The Foley self-retaining bag catheter has eliminated practically all of the troublesome features that formerly attended constant drainage with a catheter held in place with adhesive tape. Some of my general practitioner friends tell me I should be cannonized for the night calls the Foley catheter has saved them during our Minnesota winters. These self-retaining bag catheters require no external fixation by adhesive tape or otherwise, can be kept in place for weeks at a time and require no adjustment or attention of any sort. In the majority of cases such a catheter is well tolerated and makes a much more satisfactory and less troublesome method of drainage than interval catheterization. When the patient is in bed or in a chair the catheter may be connected to a drainage bottle. However the catheter may be clamped off and permit the patient to be up and about with considerable resumption of activity. At intervals the catheter is opened to empty the bladder.

In cases of very long standing obstruction accompanied by large residual urine the degree of renal function impairment may be severe and recovery of function very slow. Several weeks or even months may be required for the maximal recovery of function possible. The management requires no specialized urologic talent. Preoperative treatment of this sort does not require confining the patient to a hospital or placing him under the care of any one but his own family physician.

The preoperative treatment just outlined may be beset by complications. The urethra may become intolerant to either interval catheterization or an indwelling catheter or there may be severe infection with chills and fever. In this event suprapubic cystostomy for drainage will be necessary. Severe infection—cystitis or ascending pyelonephritis may require hospitalization. Daily bladder lavages with antiseptic solutions such as 1-2000 silver nitrate or 1-10,000 Zephiran or bladder instillations of 2 per cent mercurochrome probably are useful in avoiding such complications. During this whole period, provided renal function is not too greatly depressed, the sulfonamides should be given in repeated courses to avoid infection, or the sulfonamides or penicillin may be used to control established infection. In a small percentage of cases epididymitis will complicate the situation. Usually it is well controlled by support and continuous hot wet applications. The latter are preferable to the ice bag in all stages of the process.

When it is apparent that improvement has reached its maximum and has come to a standstill nothing will be gained by further postponement of operation.

The choice of operation—transurethral resection, suprapubic prostatectomy, or perineal prostatectomy—must lie with the surgeon or urologist who does the operation. The general practitioner or family physician—or for that matter the patient himself—may have a preference for any one of the three procedures and hope that it may be employed. This preference and hope can be expressed intelligently only as a preference for a particular surgeon or urologist particularly talented and skilled in the preferred procedure.

This business of the choice of operation and choice of surgeon or urologist is a touchy business. If I had more tact and less temerity, I probably would say nothing more about it.

Although the choice of operation is not a part of the general practitioner's role in the management of vesical neck obstruction it is his job to refer the patient to a surgeon or urologist. And so some comment anent the matter may be to good purpose.

In any case of vesical neck obstruction, it is desirable to employ the method of operation that is best suited to the patient and to his particular form of obstruction. To employ the operation that is best suited to the doctor is still more important. If the surgeon who does the job is not qualified to do it properly it will not help much to choose the operation best suited to the patient. Both patient and doctor will be better off if the doctor employs always the particular operation best suited to his talent, and to his ability to perform it properly, even though it is not academically the operation best suited to the patient and his particular form of obstructive deformity. Certainly mortality, morbidity and general results will be best served if this consideration dictates the choice of operation.

Only if the surgeon's diversity of talent permits may the operation be suited to the patient.

Three methods of operation for vesical neck obstruction are available: suprapubic prostatectomy, perineal prostatectomy and transurethral resection. Each one of the three makes a different demand upon skill, ability, training and experience.

Suprapubic prostatectomy makes the least demand on these. It is a relatively foolproof procedure. Combined with some degree of general surgical ability and some experience in the procedure itself, it is capable of reasonably good execution in hands of average skill.

A well performed suprapubic prostatectomy yields an excellent result. Faults in its performance may increase mortality but the technic is so simple there is little opportunity for technical faults to give a poor result.

Perineal prostatectomy is a tricky and treacherous procedure. It requires intimate knowledge and familiarity with perineal anatomy. It is not just recognition of structures exposed and seen; it is knowing where they are and how not to see them—the lumen of the rectum for instance.

The really significant merit of perineal prostatectomy is its low mortality. For some reason there is no shock and it is peculiarly devoid of the fatal complications that go with the suprapubic operation.

Any surgeon can perform a perineal prostatectomy and no matter how badly it is done or how poor the result, the patient usually recovers. What surgeon is able to do a perineal prostatectomy properly and regularly secure a good functional result is another question.

Transurethral resection makes by far the greatest demand on special skill, training and experience. It is a highly technical procedure in which many factors are concerned and determine competence.

All suprapubic and perineal operations are much alike. Any one competent to do them at all is competent to do them in all cases. This is not the case with resection. Depending on gland size, length of urethra, configuration of deformity, vascularity of tissue and many other factors, resection varies from a relatively simple and easy procedure to one of great complexity and difficulty that will tax the ultimate in skill and resourcefulness.

This fact makes competence as a resectionist dependent on another, somewhat ethereal factor which applies little if at all to the open operations. This factor is the individual's own honest and accurate appraisal of his ability as a resectionist and willingness, despite the patient's clamour, to limit the procedure to cases that come within the scope of his ability.

In view of all this, it is hard to understand why good old reliable suprapubic prostatectomy is replaced by a new and difficult procedure forced into hands where it does not belong.

I submit that in the choice of operation for vesical obstruction, the first and most important thing is to suit the operation to the doctor. When that has been done and depending on the doctor's talent and ability in each of the three procedures, the operation may be fitted to the patient.

Only the surgeon himself knows what his skill may be with each of the three procedures. In view of that the family physician never should prescribe the form of operation to be done.

In the hands of the surgeon who is thoroughly competent to do all three operations and is not prejudiced, transurethral resection will be the operation of choice in 85 to 90 per cent of cases, suprapubic prostatectomy in about 10 per cent and perineal prostatectomy in the remainder.

Such a surgeon or urologist will reserve the suprapubic and perineal operations for cases presenting a degree of technical difficulty to resection. Glands of very large size, abnormally vascular ones, and great elongation of the prostatic urethra make for such difficulty. Among these cases such a surgeon will have a preference for the suprapubic operation because of the comparatively poorer promise of a good result by perineal prostatectomy in his hands. However if the patient is very old and decrepit, and a poor surgical risk, perineal prostatectomy will be the operation of choice because of its greater safety.

The surgeon who is fair to his patient and to himself, will limit his use of these three operations according to his ability to perform them. Always he will employ the operation which best combines with his particular skill and ability to serve best the welfare of the patient in his hands—whether it be by the suprapubic, perineal or transurethral method.

If we recognize this, and are governed accordingly, prostatic surgery will be perfectly individualized in respect to both patient and surgeon, and will best serve the welfare of patient, doctor and medical science. Now about postoperative and follow-up treatment. These patients are not fully recovered at the time of discharge from the hospital. To postpone discharge until full recovery would be a needless loss of time and impose unnecessary expense. The patient needs routine follow-up observation and care.

What the end result is to be is never known at the time of discharge. The chief promise of a good eventual end result is absence of residual urine. The presence of some little residual for some weeks following operation, particularly transurethral resection, is of no grave moment. If the operation has been adequate the residual will disappear. So check up on your urologist by frequent residual determinations. If at the end of six or eight weeks residual in excess of 30 c.c. continues the patient should be sent back for check-up cystourethroscopic examination.

In some cases persisting residual urine is due to bladder atony and may continue even though the obstruction has been completely relieved. In such cases Mecholyl bromide or the more recent preparation Furmethide may be effective in increasing bladder tone and promoting complete emptying. Putting the bladder at rest for a few days with an indwelling catheter along with these drugs is a good plan.

Following any operation for vesical neck obstruction there is always pyuria. It will continue until the surface of resection or enucleation has completely healed and covered with epithelium. Until that has happened there is no use in trying to clear the pyuria. At the end of six weeks with enucleations, longer with resections, sulfonamides, lavages and other means of clearing the pyuria should be started if pus in the urine continues.

Alkaline encrustation of the surface of resection or enucleation may occur during the repair process. This is due to infection with urea splitting organisms and causes an alkaline urine. Large doses of acidulating drugs such as ammonium chloride are helpful in avoiding this complication. Bladder irrigations with diluted phosphoric acid are to the same purpose.

Epididymitis and other complications need no special comment.

CONCLUSION

It may be said that the management of vesical neck obstruction should be the joint enterprise of the family physician and the urologist. In the past the general practitioner has not fully played his part—either because he has deliberately avoided his proper part or because the urologist has deliberately taken it from him.

During the past few years my general practitioner colleagues and I have managed the matter in the way indicated with very happy results. It has had the effect of greatly shortening the period of hospitalization and has diminished expense. It has proved to be a small part of the answer to the cry against the prohibitive cost of medical and surgical care.

BODY SNATCHERS*

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The term "resurrection days" is applied to that period when the Irish, the Scotch, and the English teachers of anatomy were compelled to deal with grave robbers. In the early part of the nineteenth century, and long prior thereto, an adequate supply of material for dissection could not be obtained, legally, in the countries mentioned. It was not until after the passage of the Warburton Anatomy Act, in 1832, that the vocation of body-snatching, which long had been regarded as a despised but necessary evil, gradually was abandoned in the United Kingdom, and in Ireland.

When and where the vocation of grave-robber arose, and the circumstances under which the members of the guild first operated are unknown. Violation of mausoleums, graves, and subterranean rooms for the dead doubtless occurred in ancient times, when gold and silver ornaments and precious gems were placed near and on the body of the deceased. Egypt may have been the birthplace for the practice. The Egyptians, as Herodotus stated, neither buried nor burned their dead. They did not bury them for fear they would be eaten by worms; they did not burn them, because they considered fire like a wild beast that devours everything it can seize. Filial piety and respect caused them to embalm the bodies, after which they were deposited, with rich ornaments and utensils, in coffins placed in spacious rooms. That such receptacles were often robbed is now well known. The robbers, however, were not interested in securing anatomic material.

Persons who disturb the dead are called "resurrectionists." In the broad sense, all of them should be regarded as lawbreakers. For, by what right does an alleged scientist of the present day disturb the tomb of the Egyptian ruler who lived 3,000 years before Christ? Or burglarize the sarcophagus of a later date? Wherein is he less sacrilegious than were the wretches who, a century ago, sold hundreds of stolen, and a few dozen of murdered, bodies to teachers of anatomy?

The desire for repose after death reaches back to early mists of the morning of the world. The primitive Christians put epitaphs on the tombs of deceased relatives, asking vengeance of Heaven on any and all who should disturb the dead. Several of the Popes hurled anathemas against the violators of the grave.

That even Shakespeare had given thought to grave-robbing vandals is evidenced by the inscription on his tomb:

"Good friend, for Jesus' sake, forbear
To dig the dust inclosed here:
Blest be the man that spares these stones,
And crust be he that moves my bones."

Avon's bard, however, has been more fortunate than were many other historic characters whose osseous tissues, to say nothing of their internal organs, have been widely distributed.

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*Read before the History of Medicine Society, Tulane University School of Medicine, January 1947.

Many persons who wielded power, changed history, and decided the fate of nations, while they were among the quick, have been accorded scandalous treatment after joining the ranks of the dead. Powerful as Cromwell had been during life, less than scant courtesy was his lot after death. On January 30, 1661, his body was dragged from its resting place and his head was placed on a spike on Westminister Hall, where it remained for about twenty-five years.

April 1, 1813, the vault containing the remains of the unfortunate Charles I was opened and the head was removed for examination.

The head of that great cardinal, Richelieu, had a similar but a more variegated fate. There is a good reason for the belief that not all of the organs of Napoleon are within his magnificent tomb. Laurence Sterne, who wrote "Tristram Shandy" and the "Sentimental Journey", ended his career in Cambridge, where his body was dissected by a professor of anatomy. Gambetta's post-mortem examination, which was made January 2, 1883, by Professor Cornil, in the presence of a coterie of distinguished savants, afforded to his enthusiastic friends the opportunity to carry off some very select parts of his anatomy.[1]

Before going into the actual story of the "body-snatchers" and who they were, I would like to say something of the history of dissection and the study of anatomy, both of which are so firmly bound up with, and directly responsible for the beginning of such a profession.

Dissection was unknown in ancient Egypt and in ancient Greece. The Egyptians often stoned the operator whose task it was to open the abdomen so that the body might be embalmed, and the Greeks inflicted the death penalty on those of their generals who, after a battle, neglected to bury or to burn the remains of the slain.

It is impossible to know whether the Greek physicians of the Hippocratic period dissected the human body.[1]

Dissection was conducted on a large scale even in the time of Aristotle, but the human body was still taboo.

Galen dissected chiefly apes and pigs.[7]

What the Greek banned in his own land, he permitted and fostered in a conquered realm. Under the rule of the Ptolemies, who were of Greek descent, human anatomy for the first time was studied systematically, practically and legally in the famous Alexandrian University. The practice of dissection of human beings did not long exist in the city of its origin, and after the second century it was unknown there.[1]

The first date of importance is 1231, when the Emperor Frederick II decreed that a human dissection should be performed at Solerno at least once in five years.[3]

When Mundinus (professor from 1306 to 1326) took the chair of anatomy at Bologna (University of Northern Italy), the study of anatomy first became popular. The bodies were usually those of condemned criminals.[7] In the year 1319 there is a record of a legal procedure there against four medical students for body-snatching, the first record that I could find of this practice.[2]

What annoyed Vesalius in his student days was the method of studying anatomy. Human cadavers were still so scarce that Vesalius found it necessary to climb gallows or rob graveyards for material.

Vesalius' book on anatomy, which came from the press in 1543, is interesting to look at in relation to the subject of "body-snatching." For example, the first page of the text opens with an O, within which we find cupids engaged in the forbidden practice of boiling a skull; behind an I the cupids, with the aid of a lighted torch, are disinterring a dead body—that is, they are acting as resurrection men.[8]

Even well into the sixteenth century dissections were not common. A member of a famous Basel family of physicians, Felix Plater, has left us in his autobiography, details of the dissections he witnessed at Montpellier between November 14, 1552, and January 10, 1557, only eleven in number. How difficult it was at that time to get subjects is shown by the risks they ran in "body-snatching" expeditions, of which he records three.[7]

The scarcity of anatomical material during this period may be further judged from the fact that Rondolet, a professor of the medical school of Montpellier, for want of other subjects, dissected the body of his own dead child before his classes.[5]

In 1629, when Rolfink at the University of Jena asked for cadavers of criminals for dissection, people were so wrought up that they hurled stones at him, and culprits before being executed implored that they not be "Rolfinked."[11]

Although dissection was legalized in various parts of the empire in the thirteenth and fourteenth centuries, no statutory requirement seems to have been made to provide subjects until, in 1387, we find that the Statutes of the University of Florence request the authorities to see to the delivery of the bodies of three alien criminals each year for this purpose. The connection between dissection and the punishment of crime was to last for 450 years.

In England and Wales the claims of anatomical dissection were recognized by the Charter given in 1540 by Henry VIII to the United Company of Barbers and Surgeons.

Scotland had been somewhat earlier in the field, the Town Council of Edinburgh having given in 1505 the Incorporation of Surgeons and Barbers "once a year a condemned man after he be dead, to make anatomy of, wherethrough we may have experience to instruct others." Although in Ireland no specific provision for anatomical subjects seems to have been made, eight persons are mentioned as Readers of Anatomy in the Charter to the Barbers, Surgeons, Apothecaries, and Periwig Makers of 1687. In France, by the middle of the seventeenth century, dissection was practiced openly.

In the British Isles, the official arrangements for dissection soon proved inadequate, and by the first half of the eighteenth century, "private anatomies" were becoming so common that it was clear that the attempts to put them down were hopeless; already the prevalence of body-snatching had led to riots in Edinburgh in 1725 and 1742.

The following is from an article in "Scots Magazine" of March 1742, reprinted in a 1942 issue of the Edinburgh Medical Journal: "For sometime past there was grounds to suspect the unjustifiable practice of stealing corpses out of their graves was becoming too common here; and on the 9th of March, a dead body was found in a house near the shop of Martin Eccles, Surgeon, (which upon inquiry was found to be one Alexander Baxter's, who had been interred in the Westkirk yard, March 2). Upon this discovery the populace was enraged, and crowded the place, threatening destruction to the Surgeons. Towards night the mob increased, (notwithstanding the precautions used by the magistrates) and having seized the Portsburgh drum, they beat to arms down the Cowgate to the foot of Niddery's wynd, till the drum was taken from them by a party of the city-guard. However, that night they broke several Surgeons' windows, and next evening forced their way into Mr. Eccles shop, though guarded by a party, and fell to demolishing everything. But the magistrates, attended by their officers of the trained bands, constables, etc., attacked and dispersed the mob, and most of them having run out at the Netherbow, that and the other gates of the city were shut, by which they were in a great measure quelled. Mr. Eccles and his apprentices were cited to stand trial before the magistrates, as accessory to the raising of dead bodies. Two of the apprentices absconded; and Mr. Eccles and the other three appeared; but no proof came out against them.

"On the 13th of March the Incorporation of Surgeons met and after having taken the above affair into their serious consideration, for their own vindication, and to testify their abhorrence of so wicked a crime, they inacted, 'That each apprentice, or servant, who shall be found accessory to the raising of dead bodies out of their graves, or, knowing the same to have been so raised, shall dissect, or witness the dissection of any such body, shall forfeit the freedom of the corporation, and all privilege competent to them by virtue of their indentures; and be immediately extruded from their master's service.—That freemen found guilty, shall be expelled from the society and they offer 100 marks for the discovery of each offender.'

"On the 15th at night, the mob, upon a rumor that George Haldane, one of the Beadles of the Westkirk, had been accessory to the raising of dead bodies, fell upon his house near the Westkirk, consisting of two stories and a garret, calling it Resurrection-hall, as if built by the gains of that unlawful traffic; and finding some pieces of old coffins, they were so exasperated, that they destroyed or carried off everything found in it. Next morning, about nine o'clock, they uncovered the house, pulled down the partitions, then set fire to the whole, and afterwards laid the walls level with the ground, without the least opposition, tho' thus employed all day. The 18th, Haldane published the following declaration in the newspaper: 'All Doctors or Surgeons in Edinburgh or about it, or within the Kingdom of Scotland, or any other person that can make it evident that I had any hand or part in lifting corpses in the West Church yard, I come in the judges hands to suffer death.' Signed George Haldane.

"The 18th, a mob entered the house of Peter Richardson, gardener at Inveresk, four miles east from Edinburgh, and burnt it, on a suspicion of his having been accessory to the raising of dead bodies in Inveresk church-yard.

"On the 26th, a street-chair with all its furniture, was, by a sentence of the magistrates, burnt at the cross by the common executioner, having some weeks before been stopt at the Netherbow port with a stoln body in it. John Drummond the chair-master and John Forsyth, the chair-carrier, deposed, that they were betrayed into this scrape, and at last impelled to take in the corpse; but notwithstanding, they were banished the city.

"P.S. Notwithstanding the troubles occasioned by the raising of dead bodies, and the beforementioned rigorous punishments inflicted by the populace, one John Samuel, Gardener at Grangegateside was detected April 6th, at night at the Potter-row port, carrying the corpse of a child that had been buried the Thursday before in Pentlow kirk-yard, whiched the waiters stopped, suspecting it to be prohibited goods. The fellow got off; but the enraged populace ran furiously to his house, and destroyed everything they found in it, except the cloaths and bedding of his wife and children, which out of compassion they gave them. Samuel absconded, but was next day apprehended, and committed prisoner to the city-jail. Upon a "fama clamosa", the mob had visited this Samuel's house immediately after demolishing Haldane's; but were prevailed upon by the neighbors not to touch it; they only warned him to be on his good behavior: but it seems the love of money made him insensible of his danger.

"(July) John Samuel has been tried before the Justiciary, for violating the sepulchres of the dead. The jury's verdict was in these words: 'We by a plurality of voices find, That the pannel, the time and place libelled, was seized with a bag and that the child so seized was Gaston Johnston, son to Robert Johnson (Wigmaker in Bristo, Edinburgh) pursuer. Find, That the grave of the said child at Pentland has been broken open, and the body carried out of the coffin; but do not find it proven that the pannel opened the said grave.' The pannel was thereupon sentenced to be whipt thro' the city Edinburgh, and banished Scotland for seven years.—The court seemed to be of the opinion that a corpse raised from the grave being found in the possession of a person, was relevant to infer his being guilty at and part of the detestable crime of violating the sepulchres of the dead; and if such low tools as the pannel were punishable, much more ought his employers. It was also observed, that by the Civil Law this crime was punished with death.—Samuel was accordingly whipt July 28; but by the care of the magistrates was protected from an insult by the populace."[10]

The revival of anatomy about 1750 under

the Hunters in London and the Munros in Edinburgh rapidly increased the number of students and still more the demand for bodies, since formal demonstration by the lecturer no longer sufficed and students carried out many dissections for themselves.

This brings us to our second period—the Golden Age of the Resurrectionists. These were of two types: the students themselves, often led by the professors' assistants; and professional body-snatchers, who worked at first singly or in pairs, but were later organized into rival gangs. The professional body-snatchers depended mainly upon disinterment, but used other tricks, such as impersonation of relatives and stealing before burial. At first prosecution was rare—indeed, the law on the subject was doubtful, and the body-snatchers themselves were convinced that they could not be indicted unless they removed the grave-clothes or a portion of the coffin.

With the increase of the traffic, more frequent and open disinterment led to public odium, and the extraordinary state of affairs is best described in the sober words of the Report of the Parliamentary Select Committee on Anatomy of 1828:

"In proportion as the public became vigilant, the laws relating to sepulture were interpreted and executed with increasing rigour; and as the price of the subjects rose with the difficulty of obtaining them, the premium for breaking the laws increased with the penalty. The exhumators increased in number, and, now being treated as criminals, became of a more desperate and degraded character. The parties of daring men who now took to raising bodies did it happen (as was frequently the case) that, while in the pursuit of the same spoil, they fell in one with another, actuated by vindictive feelings, and regardless of the caution and secrecy in which the successful continuance of their hazardous occupation must depend, had contests in the places of sepulture—left the graves open to public gaze, or gave information to magistrates, or the relatives of the disinterred, against their rivals. The public was excited against the teachers of anatomy by these outrageous acts; 'and this to such a degree, that of late, in many cases, individuals, out of solicitude to guard the dead, have taken upon themselves to dispense with the laws of their country, and have fired upon parties attempting disinterment.' "[3]

These criminals, as they now were, also were known by the various names of fishermen, body-snatchers, sack-'em-up men, and resurrectionists. These resurrection-men became absolutely indispensable to the anatomist, for they were practically the only source from which subjects could be procured.

If a resurrectionist was imprisoned, the anatomist helped him and supported his family. If an anatomist refused to traffic with them, or protested at their frequent extortions, not only was he deprived of material, but he was marked for vengeance. The stronger gangs, by intimidation and bribery, by employing sextons, watchmen, grave-diggers, and undertakers, monopolized the cemeteries, and dictated terms to the men of science. Corrigan, who described the water-hammer pulse, thus summed up the situation: "The absurdity still existed that while the law punished any one found procuring a dead body for dissection; the educational laws required that every candidate should possess a practical knowledge of it." In the early nineteenth century, Englishmen could become physicians and surgeons only by violating the law.

Another oddity of this law consisted in the provision that to steal a dead body constituted a misdemeanor, but to steal an inch of the dead body's grave-clothes meant a felony; naturally the resurrectionist left the grave-clothes behind in the coffin, though on rare occasions haste made them careless. For example, the well-known body-snatchers, Vaughan and his wife, after attending two funerals in one day at Plymouth, exhumed the bodies at night-fall. Detected, they were sentenced to one month's imprisonment; they were then tried for a stocking which had clung to one of the dead legs, and were transported for seven years.

The resurrection-men boldly entered work-houses and poor hospitals, claiming to be relatives of the recently deceased; usually they secured their prey, for the authorities welcomed the opportunity of saving the expense of a pauper's funeral. At times they sold a body to one anatomist, stole it from him, and resold it to another; at other times they delivered drunkards, instead of subjects, in their sacks, and pocketed their pay before the deception was discovered. A gang would demand a large bonus from a school on the grounds that they would not supply material to any other school, and by this device received bonuses from every school. The resurrectionist suffered far more from rival resurrectionists than from the police. They were men of skill and cunning, and as restrictions tightened, the price of the cadaver increased. In the season when there was no dissection in England, they traveled to France or Spain in the wake of the armies; after a battle they extracted the teeth of the fallen soldiers, and realized fortunes from the London dentists. The resurrectionist, Butler, upon being asked where he got his teeth, replied, "Oh, sir, only let there be a battle, and there'll be no want of teeth. I'll draw them as fast as the men are knocked down."[8]

The laconic diary of a London anatomist in 1811, who had the assistance of two "professionals" called Jack and Butler, shows the wholesale way in which resurrectionists worked. "Got *six*, packed *three* for Edinburgh, *one* to Guy's. January 15, Packed *two large, one small* for Edinburgh. Jack and Butler drunk as before." Jack (Harnett) generally worked with his friend, Crouch, and these two combined the accomplishment of body-lifting with that of horse and teeth stealing, amassing thus 6,000 pounds.[9]

A resurrectionist—probably the notorious Ben Crouch—admitted to having made a complete corner in the supply for London between 1809 and 1813. According to his book, in 1809, "the number (of bodies) in London was 305 adults and 44 small subjects under three feet; but the same year there were 37 for Edinburgh and 18 we had on hand that were never used at all."[3]

An illustration of the hate that the general populace had for these men is recorded in Darwin's autobiography in which he says:

"I once saw in the streets of Cambridge almost as horrid a scene as could have been witnessed during the French Revolution. Two body-snatchers had been arrested, and while being taken to prison had been torn from the constable by a crowd of the roughest men, who dragged them by their legs along the muddy and stony road. They were covered from head to foot with mud, and their faces were bleeding either from having been kicked or from the stones; they looked like corpses, but the crowd was so dense that I got only a few glimpses of the wretched creatures."

The dexterity of the resurrection-men with crowbar and chain was nothing short of marvelous. With utmost speed, without a sound they removed a body in the darkness, put back the death-clothes, and restored the grave so skillfully that many flowers were planted and thousands of tears were shed over empty coffins.

Sir Astley Cooper informed the Committee of the House of Commons on one occasion that the anatomical teachers of England were entirely "at the feet of the resurrection-men." It was not pleasant to be at the feet of such men, because they were not gentlemen: for example, a typical resurrectionist, the tall, pale-faced Merrilees, sold his sister to the surgeons.[8]

To protect the dead, many plans had been tried, which only briefly can be mentioned here. Well-to-do persons hired watchers with loaded blunderbusses at 6 d (pennies) a night, with free liquor, to guard the graves of relatives until putrefaction was too far advanced for the bodies to be worth exhuming, and iron cages to prevent exhumation still exist in some of the graveyards more than fifty miles from Edinburg.[3]

Much importance was placed on the use of spring-guns and iron coffins, and in an advertisement printed October 13, 1822, we learn that "the only safe coffin is Bridgman's Patent Wrought iron one, charged the same price as a wooden one, and is a superior substitute for lead." There was another maker of patent coffins, who is mentioned by Southey in his ballad called "The Surgeon's Warning." The ballad shows the fear of a dying surgeon, lest his apprentices should serve him after death, as he, during his life, had served other persons:

"And my 'prentices will surely come
And carve me bone from bone,
And I, who have rifled the dead man's grave,
Shall never rest in my own.

Bury me in lead when I am dead,
My brethren, I entreat,
And see the coffin weigh'd I beg,
Lest the plumber should be a cheat."

The surgeon's wishes were duly carried out as regards his coffin; money was given to watchers to keep guard over the grave. The " 'prentices", however, were able to buy the watchers, and so:

"They burst the patent coffin first,
And then cut through the lead,
And they laughed aloud when they saw the shroud,
Because they had got at the dead.

And they allow's the sexton the shroud
And they put the coffin back,
And nose and knees they did squeeze,
The surgeon in a sack."

Here is the final verse:

"So they carried the sack pick-a-back,
And they carved him bone from bone,
But what became of the surgeon's soul,
Was never to mortal known."

That all protective devices at times were useless is evidenced by the testimony which was given before the House of Commons (Select Committee on Anatomy) in London 1828, by Sir Astley Cooper, who often paid huge sums to the Resurrection-men. Of Sir Astley it may truly be said, that no man knew so much of the habits of the crimes, and of the few, if any, good works of the "sack-'em-up" men.[1]

By 1820 the matter had become a public scandal. In 1828, of 800 students in London, about 500 actually dissected. Since the optimum number of subjects per student was said to be three—two for dissection and one for operative surgery—about 1,500 bodies were annually required. The price per body was eight to ten guineas (one guinea equals 21 shillings or about five dollars), and had been as high as 16 guineas, as compared with one or two guineas 30 years previously. The state of public opinion was such that, according to Sir Astley Cooper, there was practically no chance of obtaining bodies from the London burial grounds by disinterment, and the chief source of supply was importation from abroad, mainly from Dublin. These might be seized by the Customs if detected, and were often so putrefied as to be unfit for use. An ingenious ex-naval captain in Dublin not only organized the export of bodies from that city but raised a public fund to pay watchers to prevent exhumation; with the fund he paid his own agents, ostensibly as watchers but actually as resurrectionists.[3]

Although Dublin was said to be a source of bodies, it is of interest that earlier in a letter written December 2, 1818 by Dr. John Cheyne, who was one of the founders of the Dublin School of Medicine and whose name is linked with that of William Stokes in "Cheyne-Stokes" respiration, the following is found:

"The bodies used in most of the dissecting rooms are deprived from the great cemetery for the poor called the Hospital Fields; these, there being no regular resurrection-men, are procured by the pupils who, fixing upon a grave, are influenced by the appearance of fresh earth scattered around it, by the freshness of the sods if there are any, and by the looseness of the substratum of soil, which is probed by a pointed iron instrument called "a poker." But notwithstanding great enterprise and exertion and disregard of danger which is sometimes also great, the work being done by amateurs, and not by regular members of this part of the profession, disappointments ensued, and the subjects raised were often in an advanced state of putrifaction and certainly the dissecting rooms are often supplied with bodies which have been under the ground for a fortnight or three weeks. In the work of exhumation the upper part of the grave is cleared, the corresponding part of the coffin is torn up, and then by means of a noose around its neck, the body is extracted by main force. The bodies tied up in a sack, neck and heels, are subject to great violence during their transport from the burying ground—as for example, they are dropped from a high wall, by which, as is well-known, fractures of the cervical vertebrae not infrequently occur."[6]

In Edinburgh the situation was even worse for that is where the famous crimes of Burke and Hare were committed.[3]

These were two Irishmen, who lived in Tanner's Close in the West Port, in Edinburgh. William Hare and Margaret Laird kept a lodging-house where a bed could be procured for a few pence; William Burke and Helen McDougal resided near by. All four drank constantly. Among Hare's lodgers, an old army pensioner, named Donald, dying before his quarterly pension arrived, owed his landlord four pounds. The parish officer came and placed Donald in a coffin. Hare wondered how to collect his debt, and talked the matter over with his friend Burke. Neither had ever been a resurrection-man, but they knew that bodies could be sold to the doctors. The parish undertaker returned and took away the coffin, which was now filled with tanner's bark, for the corpse of Donald was lying in Hare's bed. Burke and Hare went to the college quadrangle, and there a student, a pupil of Robert Knox, directed the pair to Number 10, Surgeon's Square. They received for it twice the amount of the debt, no questions were asked, and they were urged to come again as soon as they had another subject. The date on which this occurred—November 29, 1827—is of deep significance in medical annals.

Another of Hare's lodgers, Joseph, the miller, lay sick with fever. He was going to die anyway, and his continued illness might be bad for the business of the house, so Burke and Hare smothered him and sold his body to Knox for ten pounds. After that, "Sold to Dr. Knox for ten pounds," became a frequent item in the accounts of the new firm. A nameless Englishman, "who used to sell spunks in Edinburgh," suffering from jaundice, was next on the list. The partners now began prowling the streets for subjects, noting the friendless men and women who haunted the neighborhood. Every week, Abigail Simpson, who sold salt and camstone came to Edinburgh: Hare and his wife invited her in for a drink, and the next day she was sold in a tea-chest. Knox was pleased with the fresh state of the body, but he asked no questions. Mrs. Hare offered several others the hospitality of her whiskey, and they went straight from Tanner's Close to Surgeon's Square. One morning Burke saw two policemen dragging a drunken creature to the watch-house. "Let her go," he said, "I know where she lives and will take her home." After sunset, Knox owned her. Burke enticed old Effie, the cinder gatherer, to Hare's stable, made her drunk, and smothered her—or as we now say, "burked her." Mrs. Hare suggested that Mrs. Burke be turned into merchandise, but Burke was too fond of his Nellie for that.

Ann McDougal, a relative of one of Nellie's other husbands was next when she came to visit Nellie. Hare commenced the stifling and Burke completed it. Burke did not want to begin first since the woman was a distant friend. Mary Haldane and her daughter, two streetwalkers, were next.

This catalog had several other entries, and the procedure was similar; one lay on the victim, and the other placed his hands over the nose and mouth—death came without leaving a mark. At times fortune favored the firm. Burke was about to bring in a forlorn old man, when he was greeted by a hearty Irishwoman, who was holding on to the hand of her grandson, a boy dumb since birth. She knew Burke, and told him she was searching for some friends, but did not know their address. As two bodies brought more than one, Burke dismissed the old man, and told the woman that "he knew the whereabouts of the folk." She followed him home, drank the proffered bottle, while the women watched the child, and when she was so drunk that she could offer no resistance, she was burked. The next morning the dumb lad showed by his actions that he was alarmed at being left alone; Burke himself said that he looked up at him with frightened imploring eyes, but Burke knew he could not cry out, and taking the dumb thing on his knee he broke his back. The tea chest was not large enough this time, and so they were stuffed into a herring-barrel and sold to Knox for 16 pounds.

Daft Jamie, a boy of 18, was next, and he too brought ten pounds. As Burke declared, "Mrs. Hare led poor Jamie in as a dumb lamb to slaughter, and as a sheep to the shearers."

Mary Paterson, a Venus of the Pavement, was next to meet up with Burke, one morning after she had spent a night in the watch-house. He invited her to his lodgings for breakfast, and Bonnie Mary, eighteen, alone and fearless, went with him. Within a few hours the body of Mary Paterson was at Surgeon's Square.

Henry Lonsdale has described the sensation produced by Bonnie Mary: "The body of the girl Paterson could not fail to attract attention by its volumptuous form and beauty; students crowded around the table on which she lay, and artists came to study a model worthy of Phidias and the best Greek art. A pupil of Knox's, who had been in her company only a few nights previously, stood aghast on observing the beautiful lais stretched in death, and ready for the scalpel of the anatomist. . . . Knox, wishing for the best illustration of the female form, had Paterson's body put in spirit, so that when he came to treat of the myological division of his course, further publicity was given to her remains."

It was on a halloween night that the firm of Hare and Burke transacted its last business. A little old lady named Docherty met Burke, was made drunk, and finally "burked." However, some other guest of Burkes, Mr. and Mrs. James Gray, became too nosey the next day and discovered the body under a straw mattress before it could be moved. They told the police of their finding even though Burke attempted to bribe them, and so finally the "jig was up."[8]

The trial of Burke and Helen McDougal began at ten o'clock in the forenoon of Wednesday, December 24, 1828. It was continued, with certain intervals for refreshment, until nearly ten o'clock the next morning. It was on Christmas morning, December 25, 1828, that the jury after an absence of fifty minutes, returned the following verdict: "The jury find the pannel, William Burke, guilty of the third charge in the indictment, and find the indictment not proven against the pannel, Helen McDougal." Burke was promptly sentenced to be hanged "upon a gibbet until he is dead, and his body to be delivered to Dr. Alexander Munro, Professor of Anatomy in the University of Edinburgh, to be by him publicly dissected and anatomized."

Hare saved his neck by becoming King's evidence, but later on his identity was discovered by some workmen in England, where he fled, and he was thrown in a vat of lime with the result that his eyes were burned out.

Burke was hanged in the presence of an enormous crowd, estimated at 37,000 persons, whose members had assembled to show their delight. On the scaffold, Burke's composure left him amid jeers, curses, and taunts of the multitude. Definite cries of "Hang Hare, too," "Where is Hare?", and "Hang Knox," were mingled with curses against Burke.

On the morning of the following day, in the presence of a select audience, Burke's body was examined in one of Dr. Munro's rooms. Early in the afternoon, complying with the sentence of the court, Alexander Munro III publicly dissected the body of Burke. It was probably the most exciting anatomic afternoon recorded in the annals of history. Burke's body was carefully dissected. His skeleton was preserved and now hangs where all may see it.

Robert Knox had his life wrecked, ruined, and embittered by the fortuitous circumstances which caused the murderers, Burke and Hare, to cross his path.

The rifling of graves had been sad enough, but now, murder had added new terrors. Each family assembled its members long before the disappearance of the sun; doors were locked with double care; working men came home at night only in groups, and even the "unfortunates" rarely were seen in the streets. All were fearful that, suddenly as well as unwillingly, they might be converted into anatomic material.[1]

The excitement created by the trial was so great that it finds reflection in the works of Walter Scott, DeQuincey, George Eliot, Mrs. Gaskell, Robert Louis Stevenson, Charles Dickens, and Robert Southey.[11]

A bill regulating Anatomy Supply was finally drawn up, but failed to pass its second reading in June of 1829.

Nothing was done for the next year until the following November when a second notorious crime completed the work of rousing public opinion which Burke and Hare had begun. On November 5, Bishop and Williams (or Head) tried to sell the body of a boy of 14 to King's College. Its appearance raised in the porter's mind a suspicion of foul play, and the Demonstrator of Anatomy detained the men with the excuse of getting change for a 50 pound note—the agreed price was nine guineas—until the police arrived. A postmortem examination showed fractures of the cervical vertebrae, and it was finally found that the body was that of an Italian boy, Carlo Farrari, who had been lured by the murderers to an empty house in Nova Scotia Gardens, drugged with opium, overpowered after a struggle, and drowned by hanging head downwards in a well. Bishop and Williams also confessed to the murder of a woman and a boy for the same purpose, and these confessions were magnified in the popular imagination until the victims of "burking" were believed to number hundreds. They were hanged on December 5, and on December 15 Mr. Warburton introduced into Commons his second Anatomy Bill, which passed, on August 1, 1832. This is the act which governs anatomical supply today in Britain.[3]

In the United States no dissection of the human body was made until 1751 when Thomas Calwalader dissected in Philadelphia. In 1765 riots repeatedly interrupted William Shippen's lectures on anatomy because of objections to his recourse to human dissection. A contemporary rhymster played upon the sentiments of mourners in his doggerel of a corpse protesting:

"The body-snatchers they have come
And made a snatch at me;
It's very hard them kind of men
Won't let a body be!
Don't go to weep upon my grave
And think that there I be;
They haven't left an atom there
Of my anatomy."

Even more violent expressions of public disapproval occurred in New York during the winter of 1788, where it is reported that the public was angered because medical students dug up bodies "not only of strangers and blacks—but the corpses of some respectable persons." What has been called the "Doctor's Mob" broke into the building where Wright Post was teaching a course in anatomy and destroyed a valuable collection of anatomical and pathological specimens. The militia had to be called out to prevent the threatened lynching of doctors imprisoned in jail for safekeeping, and three members of the crowd were killed. About the same time outbreaks against dissection occurred in Baltimore. Other similar attacks occurred in the United States, and in 1844 the equipment of the college in St. Louis was destroyed.[11]

Dr. Windle, speaking at a Founder's Day Address in 1943 for the Northwestern University Medical School, has this to say about early dissection and anatomy at that Medical School:

"The practical work in anatomy was conducted in the dissecting room, and at that time after dark, a reminder of the day when human dissection was conducted surreptitiously if at all. The professor of anatomy was assisted by a demonstrator of anatomy. The duties of the demonstrator were fully as essential as those of the professor, for it was he who procured the material for dissection in addition to making the dissections for demonstrations. Obtaining cadavers was a nefarious and dangerous task in 1859. There were no laws in this part of the country to make possible the use of unclaimed dead for anatomical purposes. None was passed in Illinois until 1833—after a great scandal in Ohio which followed discovery of the body of a son of a former President of the United States, shorn of patriarchal beard and hanging by the neck in an elevator shaft of a medical college. Medical assistants in those days really earned their salt, in fact, they risked their lives for the good of their profession. Only a few years before, at St. Charles, Illinois, one was killed and the professor of anatomy wounded by a mob bent upon recovering the body of a fellow townsman. The professor's house, with bullet holes in the door, still stands in St. Charles."[12]

The imperative need for scientific medical investigation by working directly with the human body through dissection is now recognized. Legislation is regulating the supply, and illicit body-snatching has therefore become unnecessary.[11]

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NEW ORLEANS

Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

THE ANNUAL MEETING

The Annual meeting of the State Medical Society was without a doubt one of the most successful meetings that the organization has had in past years. Part of the success of the meeting may be attributed to the visitation of some six well known out of state physicians which was made possible by the action of the Executive Committee. The six visiting physicians all gave very well worthwhile presentations. The opportunity given to the members of the Organization to talk to them after their formal papers was a splendid chance to meet some of the medical leaders of this country.

The attendance was quite good, slightly under 800 people registered, of whom 174 were from out of New Orleans.

The meeting was marred by the unfortunate absence of President McMahon, who was recovering from an emergency appendiceal operation. We are glad to say that Dr. McMahon is now well on the road to recovery, nevertheless the members missed this outstanding, always pleasant and agreeable member of the Society who held the highest honor that the Society can confer on one of its members.

The various actions taken by the House of Delegates will be reported in the organization section of the Journal.

It might be added that we are indeed pleased to note that the next meeting of the organization will be held in Monroe whose physicians have a reputation for hospitality second to no other group in the State.

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NEW OFFICERS

The House of Delegates in their selection of officers, committees and delegates and alternates to the American Medical Association has chosen a group of physicians who are outstanding in organized medicine. The Journal wishes to congratulate the House of Delegates upon its selections and at the same time extend its felicitations to the new officers who will be called upon to serve the State Society.

For the coming year Dr. Gilbert C. Andrson of course, will be the President. Dr. Anderson has been very active in organized medicine as well as on various charitable boards and similar activities in the city. Dr. Anderson is one of the faculty of the Louisiana State University Medical School and is a past president of the Orleans Parish Medical Society.

The new President-elect, Dr. M. D. Hargrove, was the unanimous choice of the House. We know of no better man who could have been selected for this position. He has participated actively in the doings of the Shreveport Medical Society and the State Medical Society. He at one time was president of the Shreveport Medical Society and Vice-Chairman of the House of Delegates of the State Medical Society. After graduating from Tulane Dr. Hargrove promptly specialized in internal medicine and has become one of the superior internists in the state.

For the position of First Vice-President Dr. P. H. Jones, former president of the Orleans Parish Medical Society, was chosen. Dr. Jones is one of the best known internists and consultants in the City of New Orleans and his reputation as a doctor is thoroughly and well established.

The Second Vice-President, Dr. Arthur D. Long, is a psychiatrist of note and fame who is at the present time the president of the East Baton Rouge Parish Medical Society.

For Third Vice-President, Dr. Charles B. Odom was the choice of the House of Delegates. A man of undoubted charm, Dr. Odom's record in the war and his activity with the Lakeshore Hospital are definite proofs that a good man was made the Third Vice-President.

We are very glad indeed that all of the Councilors whose term of office had expired were re-elected. Dr. Guy Jones, of the Third District, Dr. W. E. Barker, Jr., of the Sixth District, Dr. C. A. Martin, of the Seventh and Dr. O. B. Owens, of the Eighth, have proved their worth and well deserved re-election.

We are also delighted to know that Dr. P. T. Talbot was re-elected as Secretary-Treasurer for a five-year term. Dr. Talbot has given unselfishly and devotedly to the interests of the Society and merits well his selection.

Dr. A. V. Friedrichs, having proved very successfully his ability as presiding officer of the House of Delegates was again made Chairman of the House and to succeed Dr. Hargrove, who had been Vice-Chairman, Dr. J. P. Sanders was unanimously elected.

The list of the various committees elected by the House will be published in the organization section. Suffice it to say that all of the members of standing committees whose terms expired at this meeting were re-elected, illustrative of the excellent work which has been accomplished by these committee members.

The delegates to the A. M. A. remain the same but Dr. A. A. Herold was added as an alternate to replace Dr. Hargrove, the President-elect.

NEOPLASTIC DISEASE ABSTRACTS

The Orleans Parish Unit and the Louisiana State Division of the American Cancer Society are sending out abstracts of recent important articles having to do with cancer and other forms of malignant disease. These abstracts are splendid and the organization is to be congratulated upon making available this service for the doctors. The abstracts are brief but contain the essential features of an article which may appear in any one of a large number of jurnals; many more than the average doctor could see or read.

These abstracts are being sent to every registered physician in the state. They can be recommended wholeheartedly for quick perusal of recent development in the field of neoplastic disease.

The headquarters of the American Cancer Society is also sending out a periodical annotated list of current cancer literature. This list contains complete references of articles in the cancer field appearing in medical journals which have appeared the previous month. Approximately one-third of these articles have brief annotations after the title of the article. It would seem that the cancer societies are making an earnest effort to acquaint physicians with the very latest information in regard to neoplastic disease.

The abstracts sent out by the state division of the American Cancer Society would seem to be very much more practical for the practitioner of medicine, whereas the bibliographical index of the American Cancer Society would be of particular value to libraries and librarians.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1947 MEETING OF HOUSE OF DELEGATES

There were many matters of importance discussed and acted upon by the House of Delegates during two sessions held at the time of the 1947 meeting of the State Society. Communications will be sent to all members and to parish societies in reference to several of these matters and it is hoped that cooperation and assistance will be given in carrying out the wishes of the House of Delegates in every manner possible. The following abstracted minutes include all action taken and approved by the general membership at a meeting held immediately following the session of the House of Delegates on May 14. The complete report of the Committee on Medical Defense is published in accordance with requirement of the By-Laws and the report of the Committee on Resolutions in accordance with recommendation contained in the report which was approved by the House.

ABSTRACTED MINUTES

ROLL CALL

First session 70 delegates, 16 officers and 10 past presidents present. Second session: 37 delegates, 12 officers and five past presidents present.

MINUTES

Minutes of 1946 meeting of House of Delegates and of Executive Committee since 1946 meeting of House of Delegates approved.

COMMUNICATIONS

State Board of Health concerning EMIC Program: Action of the House of Delegates and Executive Committee was reaffirmed and a committee is to be appointed to meet with the President of the State Board of Health to ascertain his reactions on this subject.

American Medical Association in re Section on General Practice of Medicine: Received and filed.

Louisiana Pharmaceutical Association in re Council on Medical, Dental, Pharmaceutical and Allied Services: President to appoint a committee to look into this matter, action to be taken after the matter is reported to the Executive Committee.

State Medical Societies in re appointment of A. W. Dent to Federal Hospital Council: House of Delegates went on record as not approving appointment on grounds of not sufficient qualifications, the secretary again to inform Mr. Warren Miller and congressmen from Louisiana of the action taken and the Executive Committee instructed to go on further, if possible, to get this appointee removed, sending copy of resolution to each senator and congressman from Louisiana.

Louisiana State Allergy Society in re organization and participation in State Society program: Motion made and carried that the allergists be given recognition.

State Chairman of the Conference of County Medical Society Officers in re meeting in Atlantic City: Received and filed.

Orleans Parish Medical Society in re notarial requirement for narcotic license: Delegates to A. M. A. instructed to do all in their power to have the A. M. A. go on record as favoring the resolution incorporated in this communication.

Citizens Committee of New Orleans in re beds for tuberculous patients; House of Delegates went on record as approving the action taken thus far by this committee.

Mr. C. P. Loranz in re inability to attend meeting: Received and filed.

Dr. Rhett McMahon in re flowers and telegram received from House of Delegates. Received and filed.

SPECIAL ORDER

Telegram and flowers sent to Dr. McMahon.

List of members who died since 1946 meeting read.

Introduction of Guests: Mrs. T. R. Tomlinson, Chairman of the Rural Health Division of the Farm Bureau and Mr. Frank Smith, representative of the Associated Medical Care Plans of the A. M. A.

Announcement by Captain Jackson of the New

Orleans Naval Air Base concerning need for medical officers for the Naval Air Reserve.

Report by Dr. C. H. Webb in re study of child health services in Louisiana being conducted in cooperation with the American Academy of Pediatrics.

Discussion was had concerning proposed bills in re compulsory medical insurance which have received endorsement of the President of the United States, however no action was taken in this regard.

There was discussion concerning right of members of the Society, who are not delegates, to attend meetings of the House. It was the opinion that all members are eligible to attend, however they can not participate in discussion unless special permission is granted. An exception to this rule is when the meeting is declared an executive session, at which time only members of the House are allowed to be present.

ACTION TAKEN

Question of additional report by the chairman of the Committee on Hospitals was discussed and motion was made and carried that the House of Delegates notify Dr. Isidore Cohn that if he has any further report to make this should be made to the next meeting of the Executive Committee.

Dr. Lester Williams was officially delegated to present the President's Medal to Dr. McMahon in Baton Rouge.

Dr. C. B. Odom appointed to represent the State Society on the Personnel Practices Committee from the State Society, the Louisiana Hospital Association and the Louisiana State Nurses' Association.

Motion was made and carried that the Louisiana State Medical Society go on record as approving the present system at Charity Hospital in New Orleans in giving preference for internships and residencies to citizens or sons and daughters of residents of the State of Louisiana, and that due notice of this action be given the press, the Board of Administrators of Charity Hospital and to Dr. O. P. Daly.

The following trustees were elected for a term of one year, from whom the Louisiana Physicians Service, Inc. will elect their directors: Drs. Rhett McMahon, G. C. Anderson, O. B. Owens, W. L. Bendel, H. W. Boggs, C. M. Horton, P. T. Talbot, J. P. Sanders, A. V. Friedrichs, J. W. Faulk, E. L. Zander, M. D. Hargrove, George Wright, W. P. D. Tilly, U. S. Hargrove and E. L. Leckert.

RECOMMENDATIONS OF THE EXECUTIVE COMMITTEE

1. That laymen be added to the Board of the Louisiana Physicians Service, Inc.—Not approved. 2. That the By-Laws be amended to provide that the Medical Defense Fund be increased to $40,000. Approved.

REPORTS OF OFFICERS AND COMMITTEES CONTAINING NO RECOMMENDATIONS

Following reports received and filed: President, Secretary-Treasurer, Chairman of Council, Councilors of First, Second, Third, Fourth, Fifth, Sixth, Seventh and Eighth Districts; Committees on Woman's Auxiliary, Aid to Indigent Members, Budget and Finance, Cancer, History of L.S.M.S., Hospitals, Industrial Health, Journal, Medical Defense, Medical Education, Medical Testimony, Mental Health, Nutrition, Public Policy and Legislation, and Tuberculosis.

REPORTS OF COMMITTEES CONTAINING RECOMMENDATIONS

Congressional: 1. That a mimeographed copy of the resumé of bills considered be sent to each member of the State Society, to the various representatives of Louisiana in the United States Congress and to the Bureau of Legal Medicine of the A.M.A.—Approved. 2. Approve representation at hearing in re S. 545 and appointment of representative left to president of the Society.—Approved.

Maternal Welfare: 1. That the State Society reaffirm its oppostion to the EMIC Plan about which at the present time there seems to be misunderstanding by the physicians of the state, and that all members of the Society be instructed, by a letter from the Secretary and by the Councilors of the various districts at component society meetings, that the State Society has discontinued the plan in the State of Louisiana, effective May, 1946, requesting members to refrain from sending in applications for maternal and pediatric care—Approved. 2. That the various suggestions in this report be carried out by the New Committee on Maternal Welfare to be appointed by the incoming president and that the House of Delegates endorse, in principle, the suggestions offered.—Approved.

Prepayment Medical Service: 1. That the State Society be requested to establish a Fee Schedule Committee to hear all suggestions and criticisms about the fee schedule.—Approved. 2. Discharge of this committee since the purpose of appointment of the committee has been accomplished.—Approved. After considerable discussion concerning the participating physician's requirement. A vote of confidence was extended the Board of Directors of the Louisiana Physicians Service, Inc. for their diligent work and their assurance to the House of Delegates that they will do everything in their power to iron out the differences which exist at the present time in this regard.

Resolutions: 1. That a copy of this report be incorporated in the minutes of this meeting and that a copy be submitted to the New Orleans Medical and Surgical Journal for publication.—Approved.

Revision of Charter, Constitution and By-Laws: The following additional amendments to the By-Laws were recommended: 1. Chapter 6, Section 1 to include "However, when there is only one nominee for an elective office the secretary shall cast the ballot for the Society". Approved. 2. Chapter

14—Insert after "Robert's Rules of Order " "Current edition".—Approved.

Rural Medical Service: 1. Formation of a Rural Health Council as follows: (1) Members: (a) Five medical members of the Committee on Rural Medical Service of the Louisiana State Medical Society. (b) Five members of the Louisiana Farm Bureau. (c) One member from the State Parent-Teacher Association who is interested in farm problems. (d) Two members from the County Agent and Extension Services. (e) One member of the Louisiana State Society for Crippled Children. (f) One member from the Louisiana Physicians Service, Inc. (g) One member from the Blue Cross plan. (h) One member from the Louisiana State Agriculture Department. (i) One member from the Louisiana Department of Education. (j) One member from the Louisiana State Hospital Association. (k) One member from the Louisiana State Department of Institutions. (l) One member from the Farmers' Home Administration. (m) One member from the Louisiana State Nurses' Association. (n) One member from the Louisiana State Dental Association. (o) One member from the Louisiana State Department of Public Welfare. (p) One member from the Louisiana Federation of Women's Clubs. (q) One member from the Louisiana Tuberculosis Association. (r) One member from the Louisiana Division of the American Cancer Society. (s) One member from the Woman's Auxiliary of the Louisiana State Medical Society. (t) One member from the Louisiana State Pharmaceutical Association. (u) Two members representing rural civic groups of the state, other than those previously mentioned. (2) Officers: To be elected from the component members of the Council and to hold office for one year and until new officers are installed. (a) President. (b) Vice-President. (c) Secretary-Treasurer. (d) Executive Committee consisting of five members, the president to act as chairman and four other members elected from the group. (3) Time and Place of meetings: (a) An annual meeting within two weeks after the Rural Health Conference meeting in Chicago, probably in February of each year. (b) Other meetings on the call of the president and one other officer or by five members of the Council. (c) Place of meeting to be rotated among the more populated towns of the state but may be in the smaller towns when voted upon by the Council as a whole. (4) Purpose of the Council: (a) To promote rural health in the State of Louisiana by whatever means the Council may see fit with approval of the Executive Committee of the State Medical Society. (5) Publicity: (a) Newspapers, special articles. (b) Special programs of the civic and other organizations interested in farm problems. Speakers to be furnished upon request largely from this Committee on Rural Medical Service. (c) Encourage interest of Parent-Teacher Associations, churches, and civic groups of the state on matters pertaining to rural health. (d) Foster closer understanding between the parish medical societies and the farm groups. (e) Magazine articles and other publicity wherever possible. —Approved. The following resolutions offered by the Committee on Rural Medical Service were also approved: 1. Be it resolved, that the Rural Health Committee be empowered to look into the changes of the state constitution relative to the participation of Louisiana in the Hill-Burton Construction Act, Public Law No. 725, and report back its findings to the Executive Committee. 2. Be it resolved, that the House of Delegates go on record endorsing a Board of General Practice by the A. M. A. and instruct its delegates to the A. M. A. Convention in June at Atlantic City, to present such resolutions at that convention. This is in confirmation of the communication from the A. M. A. relative to the organization of a section of general practice. 3. Be it resolved, that the Rural Health Committee be instructed to work with the Tulane and L. S. U. Medical Schools and the hospitals of the State of Louisiana inaugurating residencies in general practice. 4. Be it resolved, that other efforts be made by the Rural Health Committee in getting well trained physicians interested in practicing in rural communities. 5. Be it resolved, that the Rural Health Committee work with farm organizations in carrying out policies toward better medical care for rural areas. 6. Be it resolved, that all parish medical societies to be requested to cooperate insofar as possible with the farm organizations and the Louisiana Rural Health Service in promoting better health for rural areas.

Scientific Work: 1. That the first part of the third paragraph of Section 3 of Chapter II of the revised By-Laws, as submitted by the Committee on Revision of the Charter, Constitution and By-Laws, be amended as follows: The Scientific Meetings shall be devoted to papers and discussion relating to scientific medicine, arranged by the Committee on Scientific Work, in cooperation with special committees appointed for this purpose, chairmen of scientific sections and the Committee on Arrangements, with approval of the Executive Committee.—Approved.

Study Rearrangement of Annual Meeting Programs: 1. That the special committee appointed to Study the Rearrangement of Annual Meeting Programs be continued until such time as the program is stabilized and the necessity for further rearrangement will not be present.—Approved.

REPORT OF COUNCIL ON MEDICAL SERVICE AND PUBLIC RELATIONS

It was stated that the two recommendations contained in the report of the Council had already been taken care of by the Budget and Finance Committee.

REPORT OF LOUISIANA PHYSICIANS SERVICE, INC.

An oral report and written financial statement of the Louisiana Physicians Service, Inc. was submitted by Dr. O. B. Owens.

AMENDMENTS TO CHARTER, CONSTITUTION AND BY-LAWS AS SUBMITTED BY COMMITTEE ON REVISION OF CHARTER, CONSTITUTION AND BY-LAWS:—Approved.

RESOLUTION APPROVED

That the Louisiana State Medical Society go on record as opposed to any reduction in isolation regulations for lepers in Louisiana, which requires 12 successive negative cultures for Hansen's Bacillus (B. Leprae) taken at monthly intervals. All treatment and research involving infectious cases be continued at the Leprosarium in Carville, Louisiana. The splendid treatment, nursing care and recreational features given inmates at the Leprosarium in Carville are to be commended. Motion was made and carried that a copy of this resolution be sent to the President of the United States, to Dr. Parran of the United States Public Health Service, senators and congressmen from Louisiana and to the press.

REPORT OF LOUISIANA STATE BOARD OF MEDICAL EXAMINERS

Report accepted and following members recommended to Governor for appointment to fill vacancy in re Dr. C. M. Horton, for a term of six years: Dr. C. C. deGravelles, Dr. C. M. Horton and Dr. W. J. Norfleet.

ELECTION OF OFFICERS, COMMITTEES AND DELEGATES AND ALTERNATES TO A. M. A.

President-elect—Dr. M. D. Hargrove, Shreveport

First Vice-President—Dr. P. H. Jones, New Orleans

Second Vice-President — Dr. Arthur D. Long, Baton Rouge

Third Vice-President—Dr. C. B. Odom, New Orleans

Secretary-Treasurer—Dr. P. T. Talbot, New Orleans

Chairman, House of Delegates—Dr. A. V. Friedrichs, New Orleans

Vice-Chairman, House of Delegates—Dr. J. P. Sanders, Shreveport

Councilor, Third District—Dr. Guy Jones, Lockport

Councilor, Sixth District—Dr. W. E. Barker, Jr., Plaquemine

Councilor, Seventh District—Dr. C. A. Martin, Welsh

Councilor, Eighth District—Dr. O. B. Owens, Alexandria.

Committee on Journal: Dr. C. G. Cole and Dr. E. L. Leckert, both of New Orleans; 3 year terms.

Committee on Medical Defense: Dr. W. A. Ellender, Houma; 3 year term.

Committee Public Policy and Legislation: Dr. Roy B. Harrison, Chairman; Dr. C. G. Cole, Dr. Gilbert C. Anderson and Dr. P. T. Talbot; all of New Orleans; Dr. O. C. Rigby, Shreveport.

Committee on Scientific Work: Dr. P. T. Talbot, Chairman; Dr. Edgar Hull; both of New Orleans; Dr. R. T. Lucas, Shreveport.

Delegate to A. M. A. 1948 and 1949—Dr. J. Q. Graves, Monroe.

Alternate to Delegate to A. M. A. 1948 and 1949 —Dr. A. A. Herold, Shreveport.

Alternate to Delegate to A. M. A. 1948—Dr. C. H. Webb, Shreveport.

PLACE OF 1948 MEETING

Invitation to hold the 1948 meeting in Monroe was accepted.

REPORT OF COMMITTEE ON MEDICAL DEFENSE

At the time of the committee report in 1946 there were six medicolegal cases involving State Society members pending. During the past year three of these expired due to five year prescription and there are now still three pending.

Since the last report suit was entered against one member for which defense was requested. The member is being defended and the suit is still pending.

In addition, legal advice was requested by a member concerning a threatened suit, however formal application for defense has not been made.

Financial reports concerning the Medical Defense Fund are on file in the office of the State Society.

REPORT OF COMMITTEE ON RESTRICTIONS

WHEREAS, the Louisiana State Medical Society is about to conclude a most interesting, instructive and entertaining meeting and

WHEREAS, arrangements for and successful handling of this meeting are due to the activity of many individuals and organizations,

IT IS DESIRED that thanks be expressed to the following:

Dr. Edwin L. Zander, Chairman of the Committee on Arrangements for his contribution of time and energy which have aided greatly in the success of the meeting.

The Orleans Parish Medical Society, host of the meeting and particularly those members who have served on committees incident to preparation for the meeting.

The Secretary-Treasurer of the State Society and his assistants for their careful attention to all details.

Dr. Donovan C. Browne, General Chairman of the scientific program for his untiring efforts in obtaining a program which has increased interest and attendance at the present meeting.

Dr. and Mrs. C. Grenes Cole and Dr. and Mrs. Roy B. Harrison for their hospitality and kindness to the officers and members of the House.

Reverend Father A. William Crandell and out-of-state guests who participated in the program.

The press for their generous allotment of space prior to and during the time of the meeting.

The Louisiana State Board of Medical E

iners for the excellent report rendered by the Secretary.

For the interest and assistance rendered by members of various special and standing committees in handling matters which have been brought to the attention of the Society during the past year.

The members of the Woman's Auxiliary of the Louisiana State Medical Society for their support in regard to plans for the meeting.

The commercial exhibitors who, in a limited space, provided exhibits worthy of review by all members present.

Companies who continued to demonstrate their loyalty to the medical profession by placing advertisements in the program.

Mr. Roy Bartlett, Convention Manager of the Roosevelt Hotel and his assistants for their attention to numerous requests for equipment and facilities.

The New Orleans Association of Commerce for assistance in arranging for satisfactory dates for the meeting and for excellent service rendered at the registration desk.

Dr. A. V. Friedrichs, Chairman of the House, for his able and efficient handling of matters considered by the delegates.

To all of the officers and members of the Society, without whose attendance the meeting could not have been a success.

We express our regret that our President, Dr. Rhett McMahon, was prevented by illness from attending this meeting and we wish to thank him for his very fine efforts and accomplishments in behalf of organized medicine and the Louisiana State Medical Society.

It is recommended that a copy of this report be incorporated in the minutes of this meeting and that a copy be submitted to the New Orleans Medical and Surgical Journal for publication.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

June 9.	Scientific Meeting, Orleans Parish Medical Society, 8 p. m.
June 10.	Orleans Parish Radiological Society, 7:30 p. m.
June 11.	Woman's Auxiliary, Orleans Club, 3 p. m. Touro Infirmary Staff, 8 p. m.
June 12.	Clinico-pathologic Conference, Touro Infirmary, 12 noon.
June 13.	Executive Committee, Hotel Dieu, 8 p. m.
June 16.	Hotel Dieu Staff, 8 p. m.
June 17.	I. C. R. R. Hospital Staff, 12:30 p. m. Charity Hospital Medical Staff, 8 p. m.
June 18.	Charity Hospital Surgical Staff, 8 p. m.
June 19.	Veterans Administration Hospital Staff, 8 p. m.
June 20.	Lakeshore Hospital Staff, 8 p. m.
June 24.	Baptist Hospital Staff, 8 p. m.
June 25.	French Hospital Staff, 8 p. m.
June 26.	Clinico-pathologic Conference, Touro Infirmary, 12 noon. DePaul Sanitarium Staff, 8 p. m.
June 27.	New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

N. J. Tessitore, Secretary.

NEWS ITEMS

Drs. H. T. Beacham, Edgar Burns, Robert Sharp, Mortimer Silvey and R. M. Willoughby attended the recent meeting of the Southeastern Section of the American Urologic Association, at Palm Beach, Florida.

Drs. B. G. Efron, A. J. McComiskey, Henry Ogden and N. F. Thiberge attended the recent meeting of the Southeastern Allergy Forum at Shreveport.

Dr. Efron was chairman of one of the roundtable discussions on hayfever.

Drs. Emmett Irwin and Thos. B. Sellers were guest speakers at the Sixth District Medical Society meeting, Tuesday, April 15, Baton Rouge.

Dr. Irwin spoke on Carcinoma of Colon; Dr. Sellers spoke on Uterine Displacements.

Dr. Howard Mahorner attended the meeting of the American Association for the Study of Goiter in Atlanta, April 3-5.

Dr. Mahorner was guest speaker before the Southern District of the Texas Medical Society, Orange, April 11.

Dr. Ogden was recently made regional secretary of the American Academy of Allergy (Committee on Public Relations).

Dr. Thomas B. Sellers presided at a panel discussion at the sectional meeting of the American College of Surgeons held in Fort Worth.

Dr. Harold Wirth was elected president designate for 1948 of the Louisiana State Dental Society at their meeting held in New Orleans, April 12.

AMERICAN AID TO FRANCE

Dr. Urban Maes has been named chairman of a Doctor's Division, to be assisted by Dr. Lucien LeDoux and Dr. Mims Gage as vice-chairmen, in the local campaign.

It is the purpose of the campaign to obtain funds for the medical center at Lorient, a French port which was largely destroyed by our bombs prior to the invasion of France.

Drive headquarters are located at International House.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

At a meeting of The New Orleans Graduate Medical Assembly held Friday, April 25, 1947 in the Hutchinson Memorial Auditorium, the following officers to serve for the coming year were installed:

Dr. Joseph S. D'Antoni, president; Dr. Waldemar R. Metz, president-elect; Dr. Dean H. Echols, first vice-president; Dr. Neal Owens, second vice-president; Dr. Maurice Lescale, third vice-president; Dr. Max M. Green, secretary; Dr. Edwin H. Lawson, treasurer; Dr. Edgar Hull, director of program; Dr. C. J. Tripoli, and Dr. C. Gordon Johnson, assistant directors of program; Dr. Hugh T. Beacham and Dr. John T. Sanders, members of the executive committee.

The following officers, elected in 1946, will also serve as members of the executive committee for the coming year: Dr. William H. Gillentine, retiring president; Dr. Lucien A. Fortier and Dr. Howard Mahorner.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LOUISIANA PEDIATRIC SOCIETY

The always active and energetic Louisiana Pediatric Society has selected for the coming year, Dr. W. C. Rivenbark, of New Orleans, as President. Dr. Bertha Wexler, of New Orleans, was selected as Secretary and Dr. Jack Strange, New Orleans, Treasurer. During the State Meeting the Pediatric Society held their annual dinner meeting at the Metairie Country Club.

LOUISIANA TUBERCULOSIS ASSOCIATION

At the annual meeting of the Louisiana Tuberculosis Association held concurrently with the State Meeting, Dr. Sydney Jacobs was selected as President for the coming year. Dr. A. A. Herold, Shreveport, and Dr. Maurice Campagna, New Orleans, were chosen Vice-Presidents. Doctors elected to the Executive Committee include Dr. J. E. Blum, Greenwell Springs, Drs. O. P. Daly, Joseph A. Danna, Edgar Hull, John H. Musser, I. L. Robbins, Suzanne Schaefer, W. L. Treuting, John M. Whitney and Julius Wilson, New Orleans.

NEW ORLEANS PURE MILK SOCIETY

At a recent meeting of the New Orleans Pure Milk Society Dr. E. A. Socola was selected as president, Dr. Maurice E. St. Martin as vice-president, Mr. Robert H. Polack as secretary-treasurer, Dr. W. C. Rivenbark as assistant secretary. The Milk Commission members and the Board members consist of a group of lay individuals and a large number of local doctors.

TOURO INFIRMARY

The regular monthly meeting of the Medical Staff of Touro Infirmary was held on Thursday, May 15 at 8 p. m. The Department of Pathology presented a clinico-pathologic conference with a discussion by Dr. Arthur Caire, III. Dr. Sol B. Stern gave a case report on cirrhosis of the liver controlled by diet. A case report on amebic vaginitis was presented by Dr. Claude Buerger, and Dr. James T. McQuitty spoke on intestinal obstruction of the small bowel.

CHARITY HOSPITAL

A meeting of the Medical Division of Charity Hospital Visiting Staff was held on Tuesday, May 20 at 8 p. m. in the Auditorium of the hospital The program consisted of the following: Case report—duodenal anomaly in a premature infant by Dr. George Austin; case report—urachal cyst in an infant by Dr. Norman Woody; review of pediatric deaths at Charity Hospital in 1942, by Drs.

R. V. Platou, A. J. Hill, J. Kometani and Norman Woody.

PLAQUE TO DR. COURET

In commemoration of 35 years of service by Dr. Maurice Couret in the Pathology Department at Hotel Dieu, a bronze plaque was presented, with appropriate ceremonies, by the Hotel Dieu medical staff to the hospital. Dr. Edmond Souchon delivered the presentation address.

Dr. Joseph S. D'Antoni appeared on the program at the annual meeting of the College of Physicians in Chicago the first part of May. Drs. George Burch and Paul Reaser presented a paper which was read at the Society of Clinical Investigation on May 5.

The Colorado Rheumatic Fever Library has been established at the University of Colorado School of Medicine in Denver. All authors of articles dealing with rheumatic fever are requested to send a reprint of their article to this library.

Dr. John H. Musser was made one of the ten Masters of the American College of Physicians at a recent meeting in Chicago.

This is a further reminder that The Third Annual Congress of Obstetrics and Gynecology will be held in St. Louis September 8-12.

A College of American Pathologists has recently been organized. The officers of the College are Dr. Frank W. Hartman, Henry Ford Hospital, Detroit, President; Dr. Granville A. Bennett, University of Illinois, Chicago, Vice-President; and Dr. Tracy B. Mallory, Massachusetts General Hospital, Boston, Secretary-Treasurer.

Dr. Ernest Lyman Stebbins, director of the school of hygiene and public health and professor of public health administration of Johns Hopkins University, has been appointed chairman of the Advisory Board on Health Services of the American Red Cross. For several years prior to joining the Johns Hopkins University staff Dr. Stebbins was professor of epidemiology at the College of Physicians and Surgeons, Columbia University.

SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE

The Southern Section of the Society for Experimental Biology and Medicine held a meeting on Friday, May 9 in room 410 of the Louisiana State University School of Medicine. The following presented papers for the scientific program: Drs. C. C. Bass, Philip M. Tiller, Jr. of Tulane Medical School; Drs. Johan T. Peters, Fred G. Brazda, Chapman Reynolds, Roland A. Coullson, Florence L. Evans and George Nelson Ronstrom from L. S. U. School of Medicine; Dr. Herbert Derman of the Veterans Administration Hospital. A business meeting of the Society was held prior to the scientific meeting.

A NEW ORGANIZATION

Preceding the A.M.A. Centennial, The American Society for the Study of Arteriosclerosis was organized in Atlantic City. Members of the Organizing Committee were: George R. Herrmann, M. D., Galveston; William B. Kountz, M. D., St. Louis; Joseph B. Wolffe, M. D., Philadelphia; Lyman G. Duff, M. D., Montreal; Harry Goldblatt, M. D., Los Angeles; W. C. Hueper, M. D., New York; Irvine H. Page, M. D., Cleveland; Robert A. Katz, M. D., New Orleans.

LOUISIANA AT THE A. M. A. MEETING

Louisiana was well represented on the program of the A. M. A. at the centennial meeting in Atlantic City, June 9-13, 1947. The following Louisiana doctors either presented papers or had scientific exhibits at this meeting. They included: Drs. G. E. Burch, Thorpe Ray, Grace A. Goldsmith, Paul Reaser, Leon J. Menville and Manuel Garcia from the Department of Medicine at Tulane; Drs. Champ Lyons and Paul T. DeCamp from the Department of Surgery at Tulane; Drs. J. Brown Farrior and Mercer Lynch from the Department of Otolaryngology; Dr. Ralph V. Platou from the Department of Pediatrics at Tulane; Drs. J. K. Howles and William B. Stewart from the Department of Dermatology of Louisiana State University School of Medicine; Drs. John Adriani and David A. Davis from the Department of Anesthesiology of Louisiana State University School of Medicine; Drs. G. H. Faget and P. T. Erickson from the U. S. Marine Hospital, Carville.

Dr. Alton Ochsner presided as Chairman of the Section on Surgery, General and Abdominal; Dr. Roy H. Turner as Vice-chairman of the Section on Internal Medicine.

BLUE SHIELD DIRECTOR SPEAKS TO LOUISIANA STATE MEDICAL SOCIETY

Speaking before the 67th Annual Session of the House of Delegates of the Louisiana State Medical Society held in the Roosevelt Hotel in New Orleans, Frank Smith, executive Director of Associated Medical Care Plans, outlined work of that national organization in organizing prepayment medical care plans on a national basis. Mr. Smith reported that at the present time 33 states now have plans providing for prepaid medical, surgical and obstetrical coverage, and 13 additional states have plans in the embryonic stage. By the end of this year it is hoped all states will have organized medical care plans to provide on a national basis a means of meeting the economic status of "low income" families in the payment of surgical and obstetrical bills, said Mr. Smith.

Associated Medical Care Plans, an affiliate of the American Medical Association, will promote "Blue Shield" prepayment insurance in behalf of the doctors of the United States to enable the people of this nation to arrange for the payment of their doctor's bill.

PAYMENT PLAN IS OFFICIALLY ENDORSED BY STATE SOCIETY

The House of Delegates of the Louisiana State Medical Society on Monday, May 12, unanimously approved the report of the Committee on Prepayment Medical Service and extended a vote of confidence to the board of directors of Louisiana Physicians Service for their diligent work and assurance to the House of Delegates that they will do everything in their power to iron out the differences which exist at the present time. Dr. O. B. Owens of Alexandria is chairman of the Prepayment Committee and president of the Plan.

The prepayment surgical and obstetrical care plan has been operating actively on a statewide basis since November 1, 1946, and is known as Louisiana Physicians Service and uses the trade name of "The Blue Shield Plan." Blue Shield is an undertaking by the Doctors of the State of Louisiana to enable the people of this state to systematically budget for paying their doctor bills.

The Blue Shield Plan in Louisiana has been granted the Seal of Acceptance of the Council on Medical Service of the American Medical Association and enjoys full membership in Associated Medical Care Plans in addition to the endorsement by the State Society.

Growth of the plan has been phenomenal. Approximately 15,000 individuals have been enrolled as members of the plan since it started and the actual premium value at the end of April was in excess of $104,000.00, according to Frank Lais, Jr., executive director of the Plan.

More than 825 physicians throughout the state have enrolled as Participating Physicians and each day additional cards are being received from the doctors acknowledging their participation. Again the request is made for every doctor to sign his participating physicians agreement and actively support the plan. Participating Physicians Agreements may be obtained by writing to the State Society or to the office of the company, Room 103, 1430 Tulane Avenue.

The Blue Shield Plan for surgical and obstetrical benefits is available to employed persons on a group basis at this time, and very shortly will be made available to individuals. The Plan as offered by the Louisiana Physicians Service is a voluntary non-profit program and is controlled by a Board of Directors selected from a Board of Trustees appointed by the Louisiana State Medical Society. The Charter provides control of the plan shall be maintained by the doctors of this state. This is your plan and it deserves your active support.

The "Doctors' Plan" provides for the prepayment of surgical and obstetrical care and includes treatment of fracture and dislocations along with certain related benefits such as diagnostic x-ray, anesthesia and laboratory services. By the payment of a small fee the employed individual may prepay the cost of his doctor's bill and through this method of cooperative participation eliminate the hazard of the catastrophic doctor's bill.

The Blue Shield Plan offers through the participating physician complete surgical and obstetrical coverage to individuals whose annual income is less than $2,000 per year and to the family group whose aggregate annual income is less than $3,000 per year. This means that approximately eight hundred and twenty-five physicians throughout Louisiana have voluntarily agreed to perform complete surgical and obstetrical services to individuals who come to them for service and whose income does not exceed the amount stated above.

For the individual whose income is in excess of the amount stated the Plan acts as an indemnity toward the doctor's bill. The Plan is available to all persons irrespective of their annual income.

The subscriber has complete freedom of choice of his physician and may use any doctor anywhere in the United States and will be paid the amount for surgical procedure rendered as provided for in the schedule of benefits. However, persons using a participating physician and whose income is below the amount stated is guaranteed complete service at no extra cost.

The Plan is operated on a state-wide basis and at this time has established offices in the cities of Baton Rouge, Alexandria, Shreveport and Monroe. The home office of the Company is in New Orleans.

"In keeping with a nation wide trend it is expected that Prepayment Medical Care Plans will provide protection for individuals and their families in need of surgical and obstetrical care without working an undue hardship upon them," said Frank Lais, Jr., executive director of the Blue Shield Plan. "At the same time, plans of this type will render unnecessary any type of federally controlled compulsory insurance for providing these benefits. "Under our Plan the patient uses his own doctor or any doctor of his choice".

HEALTH OF NEW ORLEANS

For the week which ended April 5 there occurred 183 deaths in the City of New Orleans which were divided 118 whites, 65 colored with 17 of these deaths taking place in children under one year of age. These figures are well above the three year median corresponding week. For the week which terminated April 12 the 142 deaths in the city were apportioned 96 white, 46 colored, with 16 deaths in children under one year of age. The following week showed approximately the same number of deaths as the previous week. There were two more white people dying this week and three less negroes. Only 11 children under one year of age were included in the total for the week. For the week which came to an end on April 26 the death rate still remained high, 158 citizens of the city and state having died in New Orleans in this particular week. Strange to say the large majority of the deaths were in the white population, 113, with only 45 of the colored people of the city expiring and with 11 infants dying. There was a distinct improvement in the week which ended May 3. Of the 127 people who were reported as dying in New Orleans this week 82 of them were white, 45 of them colored but 13 were infants. For the week which was finished May 10 there were listed 120 deaths in the city, divided 73 white, 47 non-white with 12 infants included in this total.

In spite of the quite marked increase in the number of deaths in the city during the month of April the year 1947 has been a healthy year. Last year in comparison there were at this time 74 more deaths in the city than this year.

INFECTIOUS DISEASES IN LOUISIANA

In the weekly morbidity report for the State of Louisiana for the week ending April 5, 1947 the following diseases were reported in numbers greater than 10: Unclassified pneumonia 86 cases, cancer 83, measles 60, influenza 49, pneumococcic pneumonia 39, hookworm infestation 34, whooping cough 28, chickenpox 23, mumps 12 and scarlet fever and diphtheria each 10. Of the somewhat rarer diseases there were two cases of poliomyelitis, one occurring in Jackson and the other in St. Helena Parish. One case of typhus fever was reported this week. There must have been a considerable epidemic of influenza for the week which ended April 12 as there were 270 cases reported this particular week. Also listed were 77 cases of pulmonary tuberculosis, 30 of chickenpox, 14 of unclassified pneumonia, 13 of measles and 11 of cancer. For the first time in many weeks there were no cases of poliomyelitis reported by the State Board of Health. The epidemic of influenza was extremely transitory as the following week which ended April 19 only 28 cases were listed. Other diseases in numbers over 10 included: 224 cases of measles, 88 of unclassified pneumonia, 53 of cancer, 47 of pulmonary tuberculosis, 35 of chickenpox, 23 of pneumococcic pneumonia and 18 of mumps. This week saw poliomyelitis again appearing with two cases developing, one in Ouachita Parish and the other in Tangipahoa. Unclassified pneumonia led all reportable diseases for the last week in April with 88 cases reported. This was followed numerically by 79 cases of measles, 77 of cancer, 30 of pulmonary tuberculosis, 21 of pneumococcic pneumonia, 19 of chickenpox and 16 of mumps. Again there were two cases of poliomyelitis reported, one from Beauregard and the other from Orleans Parish. The first week in May saw unclassified pneumonia still rampant with 67 instances. This, however, was exceeded by 89 cases of cancer which were reported this week. The following diseases were also listed: Forty-five instances of measles, 43 of pulmonary tuberculosis, 17 of pneumococcic pneumonia and 13 of chickenpox. Not a single case of poliomyelitis was reported this week although there were three cases of meningococcic meningitis.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending March 31, 1947

	Total This Month	Total Previous Months	Total To Date 1946
Chancroid	63	115	178
Gonorrhea	1302	2498	3800
Granuloma inguinale	20	18	38
Lymphopathia venereum	7	19	26
Syphilis	1230	1810	3040

PASSANO AWARD

Selection of Dr. Selman A. Waksman, Microbiologist at the New Jersey Agricultural Experiment Station, New Brunswick, New Jersey, as the 1947 recipient of the Passano Foundation Award is announced by the Board of Directors of the Foundation. Presentation of the $5000 cash award will be made at a dinner to be attended by about 100 outstanding medical men at the Ritz-Carlton Hotel, Atlantic City, on Thursday evening, June 12.

Sir Howard Florey, of Oxford, England, who was knighted for his development of the clinical applications of penicillin, will make a brief address, following which the award will be presented to Dr. Waksman by Robert S. Gill, President of The Williams & Wilkins Company and of the Passano Foundation. Dr. Waksman's address will be entitled, "Antibiotics and Tuberculosis—a Microbiogical Approach".

Dr. Waksman receives the award for his original research in the field of antibiotics culminating with his discovery of streptomycin, which among its many uses gives promise of being useful in the successful treatment of tuberculosis. Previous winners of the Passano Award are: Dr. Edwin J. John, of Harvard Medical School, and Dr. Ernest W. Goodpasture, of Vanderbilt University Medical School.

WAR DEPARTMENT PROFESSIONAL MEDICAL FILMS

Lt. Colonel George H. Lord, Signal Corps, Headquarters of the Fourth Army, Fort Sam Houston, Texas, has a very considerable number of films prepared by the War Department which are available for medical society meetings. These films for the most part deal with war conditions. If medical societies are interested in showing these films information may be obtained from Colonel Lord as to those that have been made and the procedure to be employed in borrowing these films.

COMMUNICATIONS

P. T. Talbot, M. D., Secretary-Treasurer
Louisiana State Medical Society
1420 Tulane Avenue
New Orleans 13, Louisiana

Dear Dr. Talbot:

Enclosed you will find copies of a letter we are required to send all fee designated doctors. We have forwarded one to all fee designated doctors, but there are quite a few members of the society whose applications are just coming in, and these have not received this letter. It will be of the utmost importance if you could emphasize the contents to all your members. Your cooperation will be appreciated.

Very truly yours,
George A. Leonard, Lt. Col. MC
Acting Chief MedicalOfficer

VETERANS ADMINISTRATION REGIONAL OFFICE

New Orleans 12, Louisiana
May 7, 1947
In Reply Refer To: 3021-10BAB

Dear Doctor:

It has come to our attention that State Pharmaceutical Associations are indicating unwillingness to renew agreements to supply prescription services because of major losses for services rendered in good faith, as a result of physicians signing their authorization legend without actually holding such authorization. Prescriptions so endorsed and filled by pharmacists are necessarily rejected by Regional Offices, resulting in financial loss to pharmacists from conditions entirely beyond their control.

To insure continuance of prescription service to veterans entitled thereto, all fee basis and designated physicians are being notified that signing the legend, "I am authorized to treat and prescribe for the above named Veterans Administration beneficiary" on prescriptions, when, in fact, an actual current authorization covering the specific service-connected disability is not held, may constitute a violation of the Federal statutes and require reference to Department of Justice for prosecution.

When it becomes necessary to prescribe for a Veterans Administration beneficiary for whom authorization has not been obtained, such prescription may be (1) mailed to the nearest Veterans Administration medical activity for filling or (2) marked "emergency" for filling locally.

In marking prescriptions "emergency", please do not sign standard authorization legend. Prescriptions marked "emergency" are not subject to filling without cost to the veteran by participating pharmacies under the terms of Pharmaceutical Association agreement. Such prescriptions, marked "emergency", if presented to private pharmacies, must be arranged for on an individual basis by the veteran, or claim for payment filled by pharmacy with the Veterans Administration if the disability for which the prescription was issued is service-connected.

This Regional Office is happy to report that, to date, we have experienced no difficulty as a result of such incidents as above, and we feel we can rely on your continued close cooperation in the future.

Very truly yours,
George A. Leonard, Lt. Col. MC
Acting Chief Medical Officer

NATIONAL RESEARCH COUNCIL DIVISION OF MEDICAL SCIENCES

Editor
New Orleans Medical and Surgical Journal
New Orleans, Louisiana

Dear Sir:

During the past ten years the Drug Addiction Committee of The National Research Council has sponsored the investigation in the laboratory and clinically of a new morphine derivative, Metopon hydrochloride (methyldihydromorphinone hydrochloride). This study has demonstrated that Metopon possesses certain outstanding advantages which could make it the drug choice for the treatment of the pain of cancer, especially in the home care of terminal cases and for that purpose the Committee has recommended its manufacture and limited marketing. The contemplated plan of limited and controlled availability of the drug is based upon its narcotic character, its somewhat limited supply on account of manufacturing difficulties, and its advantageous applicability only to the type of case indicated. The manufacturers, Mallinckrodt Chemical Works, Merck & Co., Inc., and New York Quinine & Chemical Works, Inc., who are the firms licensed to manufacture morphine, and the distributing pharmaceutical houses, Sharp and Dohme, Inc., and Parke, Davis & Co., have agreed not to advertise the compound, but to leave its introduction to the profession entirely in the hands of the Drug Addiction Committee. We believe the best method of introduction to be publication of a clear statement of the drug's properties as nearly simultaneously as possible in the national and state medical society journals and to this end the Committee asks your cooperation.

Sincerely,
Nathan B. Eddy, M. D., Sec'y.
Committee on Drug Addiction

METOPON HYDROCHLORIDE

In 1929 with the funds provided by the Rockefeller Foundation the National Research Council, through its Committee on Drug Addiction, undertook a coordinated program to study drug addiction and research for a non-addicting analgesic comparable to morphine. Metopon is one of the many compounds made and studied in this coordinated effort.

The high analgesic effectiveness of oral doses (with the elimination of the disadvantage to the patient of hypodermic injection), the absence of nausea and vomiting even in patients who vomit with morphine or other derivatives, the absence of mental dullness and the slow development of tolerance and dependence place Metopon in a class by itself for the treatment of the chronic suffering of malignancies, and it is for that purpose exclusively that it is being manufactured and marketed.

Metopon will be avaiable *only* in capsule form *for oral administration.* The capsules will be put up in bottles of 100 and each capsule will contain 3.0 mgm. of Metopon hydrochloride. They can be obtained by physicians only from Sharp & Dohme or Parke, Davis & Co., on a regular official Narcotic Order Form, which must be accompanied by a signed statement supplying information as to the number of patients to be treated and the diagnosis of each. The drug will be distributed for *no other purpose* than oral administration for chronic pain relief in cancer cases.

The dose of Metopon hydrochloride is 6.0 to 9.0 mgm. (2 or 3 capsules), to be *repeated only on recurrence of pain,* avoiding regular by-the-clock administration. As with morphine, it is most desirable to keep the dose at the lowest level compatible with adequate pain relief. Therefore, administration should be started with two capsules per dose, increasing to three only if the analgesic effect is insufficient.

Tolerance to Metopon hydrochloride develops slowly. It can be delayed or interrupted entirely by withholding the drug occasionally for 12 hours or for as much of that period as the incidence of pain will permit.

To each physician will be sent a record card for each patient to whom Metopon hydrochloride is to be administered. He will be requested to fill out these cards and return them in the addressed return envelope. He must furnish this record of his patient and his use of Metopon hydrochloride if he wishes to repeat his order of the drug. The principal object of this detailed report is to check the satisfactoriness of Metopon hydrochloride administration in general practice. *The physician's cooperation in making it as complete as possible is earnestly solicited.*

WOMAN'S AUXILIARY

MRS. J. W. WARREN HONORED AT TEA

On Wednesday, May 14, 1947, Mrs. J. W. Warren, the new president of the Woman's Auxiliary to the Louisiana State Medical Society, was honored with a Tea given at the Orleans Club in New Orleans.

Members and delegates were received by Mrs. J. W. Warren, Mrs. Joseph S. D'Antoni, President Orleans Parish Auxiliary, Mrs. Arthur D. Long, Jr., Past President, Mrs. O. B. Owens, President-elect, Mrs. Edwin Zander, Mrs. Ashton Thomas and Mrs. Dan J. Murphy. Mrs. C. Grenes Cole was Chairman of Arrangements and Mrs. Paul G. Lacroix and Mrs. Carroll Gelbke poured during the Tea hour.

As an added attraction Mrs. Warren, members and delegates were entertained in the Ball Room of the Orleans Club with a Ballet which immediately preceded the Tea.

HONORED GUESTS ATTEND PAST PRESIDENT'S LUNCHEON

Mrs. Arthur D. Long, Jr., Past President of the Woman's Auxiliary to the Louisiana State Medical Society, was honored at a Luncheon held at the Metairie Country Club in New Orleans at 12:30 p. m. on Tuesday, May 14, 1947.

With Mrs. Long at the Speakers' table sat honored guests Mrs. Jesse D. Hamer, President, Woman's Auxiliary to the American Medical Association, Mrs. Wiley R. Buffington, President, Woman's Auxiliary to the Southern Medical Association, Mrs. J. W. Warren, President, Woman's Auxiliary to the Louisiana State Medical Society, Mrs. O. B. Owens, President-Elect, Woman's Auxiliary to the Louisiana State Medical Society, Mrs. Joseph S. D'Antoni, President, Orleans Parish Medical Auxiliary and Mrs. Boni J. DeLaureal, Toastmistress.

Mrs. Hamer addressed the group and encouraged all members to attend the Convention of the Woman's Auxiliary of the A. M. A. to be held in Atlantic City in June. Mrs. Hamer in her interesting talk listed four aims of the American Medical Association. 1—That we back Hygeia, as it is the only authentic health magazine sponsored by the American Medical Association, 2—that we help on any endeavor sponsored by the American Medical Association Health Program, 3—suggested enlisting aid and support in nurse recruitment, and 4—assist with the new project of Romance and History of Medicine.

Mrs. Wiley R. Buffington, President of the Southern Medical Association, extended an invitation to the group to attend their next meeting in Baltimore and stated that the Southern Medical

Association has a fund to help deserving students through medical school.

Honor was paid to the ladies responsible for entertainment during the Convention period who were: Mrs. Joseph S. D'Antoni, President of Orleans Parish Medical Society; Mrs. Ashton Thomas, wife of the President of the Orleans Parish Medical Society; Mrs. Carroll Gelbke, Registration; Mrs. C. Grenes Cole, Information; Mrs. W. C. Rivenbark, Publicity; Mrs. Carl Wahl, Telephone; Mrs. H. F. Brewster, Transportation; Mrs. Jerome Landry, Tickets; Mrs. Anees Mogabgab, Flowers; Mrs. George J. Taquino, Entertainment; Mrs. Aynaud Hebert, Tea Girls; Mrs. H. Theodore Simon, Printing; Mrs. Sidney L. Charbonnet, Jr., Pages; Mrs. Paul Lacroix, Exhibits; Mrs. M. T. Van Studdiford, Finance.

PRESIDENT'S ANNUAL REPORT

As President of the Woman's Auxiliary to the Louisiana State Medical Society, I beg to submit the following report of 1946-47:

An attempt was made this year to define and clarify the duties of the Program and the Public Relations chairmen, and to demonstrate the telescoping of their activities to form a better unified Auxiliary program. Each Program chairman was urged to make room for the inclusion of the work of all other chairmen, including Public Relations, Legislation, Hygeia, Cancer Control, etc., in her program planning. Since particular stress was laid on the new project undertaken by the Auxiliary this year, the *EXTENSION OF MEDICAL SERVICES THROUGH VOLUNTARY PREPAYMENT PLANS*, all chairmen focused their attention on different phases of this subject, with gratifying results.

While the legislative chairmen kept the groups posted and alert to any new developments in Washington, the Public Relations chairman bombarded the Auxiliary with leaflets on every aspect of the trend to socialized medicine that she could find. She distributed the following which were used as study material and often passed along to debating teams: *VOLUNTARY HEALTH INSURANCE V. COMPULSORY SICKNESS INSURANCE; THE DOCTOR GLARES AT STATE MEDICINE; A DOCTOR LOOKS AT THE WAGNER-MURRAY-DINGELL BILL; THE PUBLIC HEALTH; THE POLITICIAN AND THE DOCTOR; CHECK AND DOUBLE CHECK; and DON'T STRANGLE YOUR MEDICINE.* A public speaker was engaged by the Louisiana State Medical Society to speak in every district in the state on the dangers in governmental control of medicine. Under the auspices of the Auxiliary this speaker reached over 500 laymen, and laid the foundation for the excellent reception the voluntary prepayment insurance plan, sponsored by the Louisiana State Medical Society, has had. This plan in Louisiana is known as *THE LOUISIANA PHYSICIANS' SERVICE, INC.* and covers surgical and obstetrical care. Study groups have been using their brochures together with New York's splendid booklet *CHECK AND DOUBLE CHECK* as texts for acquainting Auxiliary members with the plan. The plan is at present available only to employees of the industrial plants and their families, and it will probably be July before individual policies can be written as the offices have not been able to care for the demand. Consequently, their offices are not too anxious for premature advertising from the Auxiliary. Nevertheless, because of the vital importance of educating ourselves and the public to the need of voluntary health insurance this aspect of our program will doubtless absorb a great deal of our time for the next several years.

The varied number of titles of monthly programs proclaims a wide scope of interest in both social activities and the most serious business of the Auxiliary. Besides book-reviews, musicals, dramatic skits, movies and travelogues, programs devoted to such topics as *SOCIAL PROBLEMS OF THE COMMUNITY, HEALTH COUNCIL, MENTAL HYGIENE, PUBLIC HEALTH, POST WAR MEDICINE, REPORT ON THE NATIONAL PEDIATRIC SURVEY, PRESENT TRENDS IN PSYCHIATRIC PRACTICE, STUDY OF THE LOUISIANA PHYSICIANS' SERVICE, INC., AIMS AND POLICIES OF THE L. S. M. S.*, lectures on *CANCER CONTROL* and *T. B.*, and articles taken from Hygeia and the Bulletin show a definite interest in community health and a growing social consciousness.

The Public Relations chairman has also called to our attention two of the A. M. A. transcripts that are currently being broadcast in Louisiana: The Melody of Life and Doctors Then and Now.

The Bulletin chairman reports 35 subscriptions.

Hygeia subscriptions have mounted to 128. Caddo Parish makes 8 gifts of subscriptions to public institutions; Ouachita gives 42 gift-subscriptions besides the 24 taken by their own members; Calcasieu sponsors a Parish-wide essay contest among all grade school pupils on topics in Hygeia.

The Organization report shows that, while two inactive groups have decided to disband, two new Auxiliaries, Tangipahoa and Jefferson Davis, have reorganized; quite a number of members-at-large have been added; and there has been an all over increase in membership, from 581 in 1946 to 693 in 1947, a gain of 112.

Other interests and activities of the Auxiliaries include working with the Girl Scouts, and on all drives such as Community Chest, March of Dimes, T. B. and Easter Seals, Red Cross, U. S. O., Cancer Control, making layettes, working on hospital guilds sponsoring blood banks, dressing centers, toy loan libraries, and collecting canned foods and medicines for European Relief.

Louisiana sponsors two of the three projects of the Woman's Auxiliary to the Southern Medical

Association. *DOCTOR'S DAY* is celebrated March 30 by most Parishes with tribute paid to the medical profession through newspaper editorials, letters, cards, flowers, donations to the State Commemoration Fund, picnics, banquets or some entertainment especially designed to honor our doctors. The Calcasieu Auxiliary donated a fund to start a library at the new Calcasieu Parish Hospital opened on Doctor's Day. Donations are made to the Jane Todd Crawford Scholarship Fund.

Other accomplishments of the Auxiliary include a revision of the By-Laws in accordance with the national model; instituting a School of Instruction for incoming officers; establishing a system of notebooks for State officers and chairmen; and setting up a membership file, showing local and state offices held by each member, as well as her affiliations with other organizations.

Louisiana is honored to have as the guests of our State Convention this year, Mrs. Jesse D. Hamer of Phoenix, the National President, and Mrs. Wiley R. Buffington of New Orleans, President of the Woman's Auxiliary to the Southern Medical Association.

The inspiration of the National officers and the splendid cooperation of the State Executive Board and the membership as a whole deserves special commendation. I should also like to express my grateful appreciation to the President and the Executive Secretary of the Louisiana State Medical Society, Dr. McMahon and Dr. Talbot, and to the members of the Advisory Board, Doctors Williams, Rand, and Spedale for their unfailing assistance, their expert guidance and counsel and their gracious courtesy in the consideration of Auxiliary problems.

Respectfully submitted,
Mrs. Arthur D. Long, President
Woman's Auxiliary to the
Louisiana State Medical Society

BOOK REVIEWS

The Care of the Aged (Geriatrics): by Malford W. Thewlis, M. D. 5th ed. C. V. Mosby Co., St. Louis, 1946. Pp. 500. Price $8.00.

This is a thoroughly revised edition of one of the earliest texts on geriatrics. A book of 500 pages from fly-leaf to flown leaf including a few blank pages must of necessity be brief in the consideration of the subject matter; yet, the data are well presented, devoid of superfluous verbiage and containing much that should prove of importance to the physician in dealing with the elderly patient. There are interesting statistical data and a few chapters devoted to such all important matters as hygienic, preventive and preclinical geriatrics.

I. L. Robbins, M. D.

La Tuberculose Renale Sous L'Angle de la Therapeutique: By Jean Cibert. Paris, Masson & Cie., 1946. Pp. 533, illus.

Renal tuberculosis is written primarily for the specialist in urology by a man who holds the title of Clinical Professor of Urology of the Faculty of Medicine at Lyon, France. The work presents a very full discussion of a single disease; over 500 pages as a matter of fact with a large number of, on the whole, excellent illustrations. The monograph should be of great value to a urologist even if he has to struggle through it with some difficulty as it is written in French. It is very doubtful if it will have an appeal to the average man because of language difficulties.

Tuberculose Renale is printed on an excellent grade of paper which makes possible a splendid reproduction of the 148 figures throughout the book. It is paper bound, as apparently are all the books which emanate from continental sources.

J. H. Musser, M. D.

Cushny's Pharmacology and Therapeutics: By Arthur Grollman, A. B., Ph. D., M. D., F. A. C. P. and Donald Slaughter, B. S., M. D. 13th Ed. Philadelphia. Lea & Febiger. 1947. Pp. 868. Price $8.50.

The first edition of this text appeared in 1899 and before the time of Cushny's death in 1926 eight editions had been published. It was further revised four times by Professor C. W. Edmunds of the University of Michigan and Professor J. A. Gunn of Oxford University, the twelfth edition appearing in 1940. Since its first appearance it has been widely used as a text in the medical schools of the United States, Canada and the British Isles. In the present revision under new authorship the general characteristics of the earlier editions have been retained.

As indicated in the preface no attempt is made to treat the subject matter exhaustively but concise, well digested accounts of the various drugs are presented in a form suitable for the undergraduate medical student and for the medical practitioner. The order of presentation or classification of drugs is with minor exceptions the same as that adopted in the eleventh and twelfth revisions. Although the form of presentation necessarily varies with the substance under consideration pertinent facts are usually given concerning the source and chemistry of the drug. This is followed by a discussion of its action on the tissues and systems of the body including the effects of toxic doses of signs of intolerance. The absorption, fate and excretion of the drug is discussed when such information is available. Finally consideration is given to therapeutic applications and the preparations with dosages of the United States and British Pharmacopoeias are listed.

In spite of the fact that sections on several new drugs have been included in this edition the book retains approximately its original size. This has been accomplished less by the exclusion of drugs not in common use than by a rewriting of the previous text with the elimination of material of little interest at the present time. Among the new drugs appearing in this edition are penicillin, streptomycin, the more recently developed sulfonamides, isonipecaine (demerol), diphenyl hydantoin sodium, tridione, thiouracil, dicumarol, synthetic estrogens, intocostrin, carbaminoylcholine, and alloxan. The discussion of the vitamins has been extended. The bibliography has been extensively revised and brought up to date. Furthermore, the recording of references in a uniform manner is a distinct improvement over previous editions. In the opinion of the reviewer this text will again be widely used in the medical schools of this country.

Ralph G. Smith, M. D.

Allergy: By Erich Urbach, M. D. and Philip M. Gottlieb, M. D. New York, Grune & Stratton, 1946. Pp. 968. Price $12.00.

The second edition of this excellent text will be welcomed by all who devote a major or minor portion of their time to the care of allergic patients. There has been rapid progress in the field of allergy since the first edition was published. The authors are to be congratulated on having so ably summarized and integrated the numerous papers that have appeared in the past four years. The book covers every phase of clinical allergy as did its predecessor and several complete sections have been added. Among these are considerations of the Rh factor, allergic bronchitis, allergic cough, eosinophilic erythredema and psychosomatic aspects of allergy. Much new material is to be found on drug allergy especially in regard to penicillin, sulfonamides and thiouracil. Additional information is collated on contact dermatitis and the complete list of concentrations used in patch testing is very helpful. One or two minor criticisms might be offered. It is open to question whether a new nomenclature embracing such terms as pathergy, metallergy, parallergy and the like is justified. There seems little point in printing the same (or practically identical) pictures twice, fig. 157 and 296, granted that it is very impressive. But these objections are trifling and the book can be highly recommended. It is very regrettable that Dr. Urbach has died but Dr. Gottlieb can be relied on to maintain in future editions the high standards set up in the first two.

Vincent Derbes, M. D.

Heparin: By J. Erik Jorpes, M. D. New York, Oxford University Press, 1946. Pp. 260. Price $6.50.

This is a splendid monograph prepared by a man who is outstanding in his experimental work with heparin. For this work he has received universal acclaim. It is very satisfactory indeed to have Jorpes' numerous contributions to this subject, plus a critical survey of the work of others in one relatively small volume.

The monograph is by no means devoted to a discussion of his own laboratory work and the work of others since the time that Howell and his collaborator McLean first published the results of their work done in Howell's laboratory some thirty years ago. The book contains, in addition, a splendid discussion on the use of heparin in clinical medicine and surgery. As a matter of fact more than half of the monograph has to do with this aspect of the use of heparin. The book therefore is and should be of value not only to the laboratorian but of tremendous importance to the clinician who may be called upon to treat, or prevent, venous thromboses, a medical problem as well as a surgical one. Actually, according to Jorpes, as complications, thrombosis and pulmonary embolism are as common among medical patients as among surgical ones.

This book represents a real contribution to scientific medicine. The format is splendid, and the excellent grade of paper that has been used by the Oxford University Press makes for easy reading and a very clear reproduction of the relatively few figures that the book contains.

J. H. Musser, M. D.

Radical Surgery in Advanced Abdominal Cancer: By Alexander Brunschwig, M. D., Chicago, The University of Chicago Press, 1947. Pp. 324, Illus. 116, tables, 16. Price $7.50.

After briefly reviewing the great epoches in the development of abdominal surgery, the author discusses the usage of the terms "operable" and "inoperable", stating: "In general, what is meant by an 'operable neoplasm' is one that can be encompassed readily by more or less standard techniques, there being no gross evidence of widespread metastases. It is inferred that the immediate prognosis is favorable. The term 'inoperable' is usually employed in connection with neoplasms for which, in the opinion of the operator, a radical procedure is not justified because of poor prognosis. This term is also used to designate the situation in which a neoplasm, possibly small and in itself resectable, has already given rise to visible spread beyond the possibility of complete excision. In the latter sense the term 'inoperable' and the evident immediate prognosis become confused, since frequently the primary growth is, indeed, resectable but there seems to be little justification for resection. The confusion of "inoperability" with "poor immediate prognosis" undoubtedly fosters not a small degree of conservatism in the surgical attack upon intra-abdominal cancer."

Developments which have occurred to expand the scope of radical surgery in advanced abdominal cancer, i. e. improved anesthesia, the more ready availability of blood and the more general employment of large transfusions, chemotherapy, and other means for better preparing and supporting patients undergoing radical abdominal surgery, are discussed. Attention is drawn to the fact that senior surgical residents in well-organized hospitals are now successfully performing many operations which, only a generation or so ago, were not attempted even by the master surgeons.

One hundred unselected consecutive operations for advanced abdominal cancer involving the stomach, small and large intestine, pancreas, liver, bile ducts, spleen, retroperitoneal space, and abdominal wall, many of which have ordinarily been considered impossible or impractical, are described. Also included in the series are reports of operations performed for intra-abdominal extension of gynecologic cancer.

Analyses of the results in respect to survival or at least temporary benefit are given, and lead the author to conclude that there is ample justification for more radical surgery in advanced abdominal cancer.

Ambrose H. Storck, M. D.

Medical Services by Government, Local, State and Federal: By Bernhard J. Stern, Ph. D. New York, The Commonwealth Fund, 1946. Pp. 172. Price, $1.50.

Among the several publications put out by the New York Academy of Medicine Committee on Medicine and the Changing Order and published through the Commonwealth Fund is this book on Medical Services by Government written by a Doctor of Philosophy who is a lecturer in sociology at Columbia University.

In the introduction one rather gets the impression that the book may represent a certain degree of propaganda for the socialization of medicine. However, the author is to be complimented upon the fact that he has made his book factual and objective and has omitted controversial points which might be subject to discussion and debate. In the initial portion of the book he presents data concerning the recent developments in medical services by state and local governments. In subsequent chapters Stern presents such subjects as Federal grants-in-aid for medical care and medical services by the Federal Government. Of the latter it might be said it is absolutely astounding to discover how much Governmental medical care is dispersed through various departments of the United States Government. The largest services of course are those rendered by the Army and Navy Medical Corps, the United States Public Health Service and the Veterans' Administration, but there are innumerable other relatively small medical services rendered to various people through the Department of the Interior, the Department of Agriculture, through the Tennessee Valley Authority and so on, ad infinitum. While unsuccessful effort has been made to organize these many small different services into the larger services, nevertheless to study this book one definitely gets the impression that there should be a Department of Health, the head of which should be in the Cabinet, which would correlate all health activities under one head.

To the Louisiana reader it is interesting to note that Louisiana contributed the highest percentage in taxes to the support of hospitals, 48.7 per cent as contrasted with 1 per cent in Alabama or with 37.8 per cent which California spends and which is the highest amount next to that spent by Louisiana for all other states in the Union. For general and other allied special medical care Louisiana spends 67.7 per cent of its total outlay, a sum which exceeds four and a half million dollars and in terms of per cent far exceeds any other state. In Louisiana the general medical care is well provided for, whereas our expenditures for service for crippled children is extremely small and for psychiatric service is considerably smaller than in most states. For the amount of money that Louisiana is spending now for medical care of its indigent it seems very doubtful that the state is in a position to afford the construction of new hospitals and new facilities. Even though the Federal Government is to finance in part the cost of construction of the buildings, these structures must be maintained and their inmates must be taken care of by the state.

J. H. Musser, M. D.

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The Commonwealth Fund, New York: Medicine in the Changing Order; Report of The New York Academy of Medicine Committe on Medicine and the Changing Order.

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University Medical Publishers, Chicago: Physician's Handbook by John Warkentin, Ph. D., M. D., and Jack D. Lange, M. D.

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